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COLOR VISION AND RECENT DEVELOPMENTS IN COLOR VISION TESTING

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THIS report is a brief, greatly simplified expression of the working hypotheses being used by color vision investigators today; a note on the ICI Coordinate System and Standard Observer; a short report on some of the developments resulting from Inter-Society Color Council (ISCC) and other activities; a notation of some typical color vision tests in current use, and an empiric classification of the types of color vision we have found in our studies.

The first two sections are simplified abstracts arranged, we hope, in a form easy to remember. The third section is really a news note and the fourth section hardly more than a short list of typical examples of color vision tests. For the fifth section we claim neither originality nor completeness. It is a practical, easily remembered outline into which we have been able satisfactorily to fit all the types of color vision so far encountered by us. We have not yet met what we thought was tetartanopia. Such a defect would probably require an addition to our classification.

I. A WORKING HYPOTHESIS FOR STUDYING COLOR VISION

There are

THREE FACTORS IN COLOR SEEING:

1. *The illuminant (ICI-C)*
2. *The eye (Standard Observer)*
3. *The object (test material)*

Confusion in color testing results from lack of standardization and specification of conditions of the test. To test one factor the other two must be held constant. No test for color vision is reliable unless the characteristics of both the illuminant and the test material are known.

From the Knapp Memorial Laboratories, Institute of Ophthalmology, Columbia University College of Physicians and Surgeons.

The illuminant is a tool used to perform a task, and good or poor performance will result from using an efficient or an ineffective tool.

The *illuminant* which should be used for all daylight color vision testing is International Commission on Illumination (I C I) Illuminant C, or its nearest practical equivalent. This illuminant is representative of overcast skylight, which has an average color temperature of about 6,700°K.

The same commission has adopted as a standard eye for providing specifications of test materials the so-called *Standard Observer* (a hypothetic observer whose color responses conform to those of the mean, statistically computed, of observers having normal color vision).

Standard test materials have not yet been adopted.

There are

THREE STANDARD ICI ILLUMINANTS:

1. *ICI-A*
2. *ICI-B*
3. *ICI-C*

The International Commission on Illumination (ICI) in 1931 adopted as international standards three illuminants, known as ICI Illuminant A, ICI Illuminant B and ICI Illuminant C. Illuminant A represents the illumination provided by a gas-filled tungsten lamp operating at a color temperature of 2,848°K (roughly a 75 watt lamp operating at rated voltage). Illuminant B approximates a color temperature of 4,800°K, which corresponds roughly to noon sunlight. Illuminant C approximates a color temperature of 6,700°K and represents average overcast skylight (average daylight).

While good color vision tests may be devised for any illuminant, most of the commonly used tests have been designed for daylight illumination and are not effective under other illuminants. Hence, ICI Illuminant C is the standard for use in daylight color testing work. The actual ICI standard being specified as a liquid filter to be used with ICI Illuminant A is inconvenient to prepare and use. The only near and practical equivalent is a Macbeth daylight lamp (tungsten lamp plus filter of Corning Daylight glass), designed to operate at approximately 6,700°K.

There are

THREE FACTORS IN THE ILLUMINANT:

1. *Quality, or spectral composition*—daylight values
2. *Distribution*—even, illuminate at 45 degree angle, view at 90 degree angle
3. *Quantity, or amount*—10 to 60 foot candles

There are three factors to be considered in the light or illuminant used to view the color test materials:

1. The quality, or spectral composition, of the light is of extreme importance. Obviously, green material cannot be correctly seen under a red light or vice versa. Most color testing is strongly influenced, and usually invalidated, by improper or haphazard choice of the illuminant. "Daylight," in the sense that it means whatever comes through the window, has but little value as a specification. For precise testing the spectral distribution of the illuminant should approximate as closely as possible that of ICI Illuminant C.

In scientific work, the one filter generally used to provide the nearest equivalent to this is Corning filter 590. This is available commercially in daylight lamps made by the Macbeth Daylighting Company.

2. The lighting should provide uniform distribution and, in order to avoid reflected glare, should fall on the test material at an angle of approximately 45 degrees, in which case the angle of view of the observer should be normal, or 90 degrees.

3. Extensive testing has shown that with other factors held constant a change in density of incident luminous flux causes no significant change between 10 and 60 foot candles. Since there is usually some stray light in the room, it is preferable to keep the general room illumination low and to use 50 to 60 foot candles from ICI Illuminant C on the test material—the higher level thus preventing interference with reliable results by small amounts of extraneous illumination from nonstandard sources, reflection from colored walls, etc.

There are

THREE FACTORS IN THE COLOR VISION MECHANISM:

1. *Proto, or first* (Helmholtz's red component)
2. *Deutero, or second* (Helmholtz's green component)
3. *Trito, or third* (Helmholtz's violet or blue component)

On the basis of the physical law of color mixing that all normal color sensations can be obtained by integrating the proper proportions of three primaries, i. e., red, green and violet or blue (additive mixtures) or by filtering from the illuminant the proper proportions of these three primaries (subtractive mixtures), it is assumed that there are three physiologic processes, each of which responds dominantly to its adequate stimulus or wavelength and all of which respond in certain proportions to all wavelengths—the integrated responses thus comprising one's color experiences.

This classification *(proto-deutero-trito)* follows naturally from the widely used terminology suggested by von Kries as a factual, nontheoretic description of the three classes of dichromasy (see page 607).

Although there are theories today which seem to satisfy all the facts of color vision (G. E. Müller, E. Q. Adams), the two best known theories are still those of Helmholtz (based on three components: a

red component, a green component and a violet or blue component) and of Hering (based on three reversible pairs of processes: red-green, yellow-blue and black-white). The Helmholtz theory is useful for classifying types of color confusions; the Hering theory is convenient in explaining color perceptions and such phenomena as after-images and peripheral field limits. Neither theory accounts for all the facts of normal and abnormal color vision, but with the use of each where it fits best and of the von Kries terminology to avoid reference to a single color theory one has a basis for classification that provides for

THREE TYPES OF COLOR VISION:

1. *Trichromatic*
2. *Dichromatic*
3. *Monochromatic*

Trichromatic color vision is the normal result of unimpeded response by all three physiologic processes in their normal proportions; dichromatic color vision occurs when there are only two differentiated processes, and monochromatic vision occurs when there is only one differentiated process.

A. There are

THREE TYPES OF TRICHROMASY:

1. *Normal color vision*
2. *Anomalous, manifesting relative deficiencies in one of the processes but requiring three primaries to match all the spectral colors*
3. *Low discrimination, manifesting poor discrimination in many or all colors but still requiring three primaries*

The definitive characteristic of the subject with trichromatic color vision is that he requires a mixture of at least three primaries to match any or all colors. *He cannot match all colors with less than three primaries.* He makes three kinds of discrimination: light-dark, yellow-blue and red-green (Hering's classification). If the amounts of the primaries he uses in making the match are normal, or nearly so, he is said to have normal color vision. If his first (*proto*) color process (*red component*) is weak, he will be relatively insensitive to red and is said to have *protanomalous trichromatic color vision.* If his second (*deutero*) color process *(green component)* is weak, he will be relatively insensitive to green and is said to have *deuteranomalous trichromatic color vision.* A rare type is the subject with *tritanomalous trichromatic color vision,* who is relatively insensitive in the third (*trito*) color process (*violet or blue component*). If he makes serious and widely scattered errors with all colors, it is assumed that all three primaries are depressed sufficiently to amount to a defect and he is classified as having *"low discrimination."*

B. There are

THREE TYPES OF DICHROMASY:

1. *Protanopia* (first process gone—formerly called "red blindness")
2. *Deuteranopia* (second process gone—formerly called "green blindness")
3. *Tritanopia* (third process gone—formerly called "violet [blue] blindness")

The distinctive feature of the subject with dichromatic color vision is that *he can match all the spectral colors with only two primaries*—hence the term "di-chromatic." He can make only two kinds of visual discrimination: light-dark and either blue-yellow or red-green, usually the former.

To the *protanopic* subject the spectrum in the extreme red appears very dark (in other words, his spectrum is shortened at the long wave end), and he finds a neutral band in the blue-green, which appears to him abnormally light. To the left of this neutral band the spectrum appears to him as yellow of varying chroma and brightness; to the right, as blue of varying chroma and brightness. The *protanopic* subject has a neutral band also in the red of the normal color system, just too purplish to be a spectral red.

To the *deuteranopic* subject the spectrum appears of about the same brightness as it does to the normal observer, and he finds a neutral band in the green. To the left of this neutral band the spectrum appears to him as yellow of varying chroma and brightness; to the right, as blue of varying chroma and brightness. The *deuteranopic* subject has a neutral band also in the red-purple of the normal color system, outside the limits of the visible spectrum.

The *tritanopic* subject finds neutral bands in the green-yellow of the spectrum and in the violet close to the extreme end of the spectrum. Between these neutral bands he sees green of varying chroma and brightness, and to the left of the neutral band in the green-yellow, red of varying chroma and brightness. This type of dichromasy is rare, and its characteristics are not as well known as are those of the other dichromatic types.

C. An observer possessing *monochromatic* vision is capable of light-dark discrimination only. He has no chromatic discrimination.

Stated in another way, it may be said that there are

THREE MAIN TYPES OF ABNORMAL COLOR VISION:

1. *Anomalous trichromasy*
2. *Dichromasy*
3. *Monochromasy*

Persons with *anomalous trichromasy* require a mixture of *three* primaries to produce all the colors they are capable of experiencing,

but they utilize anomalous proportions of the primaries. The two more common types of this defect are the *protanomalous* (predominantly red weak) and the *deuteranomalous* (predominantly green weak). A rare type is the *tritanomalous* (predominantly violet weak). Like the person with normal trichromasy, the seeing of persons with anomalous trichromasy includes light-dark, blue-yellow and red-green discriminations.

Persons with *dichromasy* are protanopic (formerly called "red blind"), deuteranopic (formerly called "green blind") or tritanopic (formerly called "violet [blue] blind"). They match all colors with *two* primaries.

Persons with *monochromasy* have no chromatic discrimination and match all colors by varying the amount (lightness or darkness) of *one* primary. They are totally color blind (achromatopsia).

There are

THREE PSYCHOPHYSICAL FACTORS IN THE COLOR OF THE TEST MATERIALS:

1. *Dominant wavelength*
2. *Luminous reflectance*
3. *Purity*

Each of these psychophysical factors must be controlled by suitable adjustment of the test material, because they have a very critical effect on the responses to the test. A change of one psychophysical factor frequently involves a change in all three attributes of the psychologic perception, although change in dominant wavelength chiefly affects the hue of the perception, change in reflectance chiefly affects the lightness, and so on. Much color testing material is worthless because of the critical influence of these three factors, since there are

THREE PSYCHOLOGIC ATTRIBUTES OF COLOR PERCEPTION:

1. *Hue*
2. *Lightness* (closely correlating with Munsell value)
3. *Saturation* (closely correlating with Munsell chroma).

A change in one psychophysical factor may change the psychologic perception of the other factors or of the test situation. The psychologic, psychophysical and physical factors are roughly correlated in the following tabulation:

Psychologic Term	Quasipsychologic Term	Psychophysical Term	Physical Term
Hue	Munsell hue	Dominant wavelength	Spectral centroid
Lightness (brightness)	Munsell value	Luminous reflectance	Area under the spectral curve
Saturation (depth of color)	Munsell chroma	Purity	Narrowness of spectral curve

II. THE ICI COORDINATE SYSTEM AND STANDARD OBSERVER

The *ICI Coordinate System* is a graphic system of plotting various colors in their relation to each other. It is based on the proportion of three imaginary primaries (red, green and blue) required by the *ICI Standard Observer* to match all wavelengths of the visible spectrum. The ICI Standard Observer, as defined in section I, is an imaginary physiologic entity whose color responses have been computed from data supplied by carefully trained normal observers. The data take the form of three columns of figures (or curves), which, when reduced to coefficients or percentages, may be plotted on the ICI Coordinate System as a curve representing the locus of all the colors of the visible spectrum. Most color stimuli used in accurate color testing are now plotted on the ICI diagram and specified in terms of ICI coordinates or are given in the Munsell color notation. (Most of the Munsell colors have been given ICI Coordinate System notations.) For color work in the United States it is usual to use ICI Illuminant C as the standard illuminant, since it represents daylight conditions. (In Great Britain it is usual to use ICI Illuminant B as the standard—noon sunlight.)

The ICI diagram is useful for studying the colors confused by an observer with defective color vision. For example, Pitt has determined the chromaticity confusions for protanopic and deuteranopic dichromatic systems and has plotted them on the ICI diagram. The zones which lie between the confusion lines shown in the diagram represent areas of the ICI diagram for a given lightness or brightness level within which the dichromatic subject makes no distinction. From zone to zone there is for him a distinguishable difference in hue or saturation or both. The chromaticity confusion lines converge to the vicinity of a point for each of the two common types of red-green blindness. Pitt has also plotted on this diagram the position of 43 colors extensively used in the paint trade. Broadly speaking, the subject with dichromatic vision can distinguish two colors only if they occur in different zones. From his diagram it is seen that the subject with deuteranopic vision can distinguish only 15 of these 43 colors, and the subject with protanopic vision, only 12. If 500 different trichromatic colors were listed, the number distinguishable to the subject with dichromatic color vision would only slightly increase and could never be greater than about 28 for the deuteranopic and 18 for the protanopic subject for a 2 degree field.

III. THE ISCC TEST DATA

For several years the Inter-Society Color Council (ISCC) has been interested in developing tests for color blindness and color aptitude. Its Color Blindness Test Committee has actively assisted in evaluating

old tests, evaluating test materials and producing new tests and new materials. The Single Judgment Test for Red-Green Discrimination, devised by the committee, is one such test. Other tests are being critically studied for their value both as screening tests (to separate observers with normal from those with defective color vision) and as diagnostic tests (to classify observers with defective color as to type and extent of defect).

Among recently devised tests are:

1. *The Farnsworth-Munsell 100-Hue Test*
2. *The Farnsworth Dichotomous Test*
3. *A simplified form of anomaloscope suitable for office use*
4. *The Hardy-Rand-Rittler Polychromatic Plates*

Typical profiles obtained with the 100-hue Test, and the Dichotomous Test have been obtained for subjects with anomalous trichromasy and subjects with dichromasy of all three types and for observers having low color discrimination.

The Rand Anomaloscope.—This is designed to give a quick determination analogous to the Rayleigh equation (the ratio lithium red: thallium green required to match sodium yellow). The anomaloscope was originally designed by Nagel to distinguish between normal trichromasy and anomalous trichromasy. It also offers a ready differentiation between protanopic and deuteranopic color vision and a diagnosis of low color discrimination for red and green. For the last determination the just noticeable difference in hue between yellow and yellow-green and between yellow and yellow-red is measured.

The Hardy-Rand-Rittler Polychromatic Plates.—These plates are designed to diagnose both type and degree of defect in color vision for both red-green and blue-yellow. Each plate consists of a white background in which are countersunk small disks of Munsell neutral gray of different sizes and values, and small disks of Munsell color of predetermined hue and different sizes and values. The color disks form geometric patterns which can be presented in different positions to provide an objective check on the color discrimination and to prevent coaching. There are four series of plates. In each plate of *series A* the patterns are presented in a given chroma of the red most difficult for the subject with the predominantly red-defective (protanopic) type and the red-purple most difficult for the subject with the predominantly green-defective (deuteranopic) type of red-green deficiency to distinguish from gray; in *series B,* in a chroma of the blue-green most difficult for the subject with the predominantly red-defective type and the green most difficult for the subject with the predominantly green-defective type of red-green deficiency to distinguish from gray; in *series C,* in a

chroma of the green-yellow most difficult for the subject with the predominantly violet-defective (tritanopic) type and the yellow most difficult for the subject with the predominantly blue-defective tetartanopic—G. E. Müller) type of blue-yellow deficiency to distinguish from gray; and in *series D,* in a chroma of the violet most difficult for the subject with the predominantly violet-defective type and the blue most difficult for the subject with the predominantly blue-defective type of blue-yellow deficiency to distinguish from gray. In the successive plates of each series the color patterns appear in increasing steps of chroma. The degree of defect is evaluated by the number of failures and their chroma distribution, and the type of defect, by the hue discriminated most poorly.

In our experimental work the results of the aforementioned tests have been compared with those given by the Ishihara Tests for Colour-Blindness (fifth, seventh and ninth editions), the Rabkin Polychromatic Plates for Testing Colour Vision and the American Optical Company's Pseudo-Isochromatic Plates for Testing Color Perception. In our opinion, the first of these tests (Ishihara) is good for screening the observer with defective red-green vision from the observer with normal vision, but it fails to differentiate adequately between protanopic and deuteranopic subjects, between protanomalous and deuteranomalous subjects and between subjects with anomalous trichromatic and those with dichromatic vision. The second of these tests (Rabkin) also can be relied on to screen the subjects with defective red-green vision from the subjects with normal color vision. In addition, it differentiates well between protanopic and deuteranopic subjects and between protanomalous and deuteranomalous subjects but does not adequately separate subjects with anomalous trichromatic vision from those with dichromatic vision. It has two plates designed to test blue-yellow deficiency, but these are not entirely adequate for the purpose. The third of the aforenamed tests (the American Optical Company's selection) comprises in one series certain of the Ishihara plates and certain of Stilling's Pseudo-Isochromatic Plates for Testing the Color Sense. The color reproductions are frequently inaccurate. Only tests of red-green deficiency are included. Like the Ishihara test, this series fails to differentiate between the different types of red-green deficiency and between anomalous trichomasy and dichromasy. However, a certain error score in a selected series of the more significant plates can be used to indicate a red-green defect. These three tests have been critically evaluated, and several reports have been published.[1]

1. Hardy, LeG. H.; Rand, G., and Rittler, M. C.: Tests for Detection and Analysis of Color Blindness: I. An Evaluation of the Ishihara Test, Arch. Ophth. **34**:295 (Oct.) 1945; II. Comparison of Editions of Ishihara Test, ibid. **35**:109 (Feb.) 1946; III. The Rabkin Test, ibid. **35**:251 (March) 1946.

IV. TYPICAL TESTS IN CURRENT USE

A. SORTING TESTS:

WOOLS—Holmgren, Jennings, Oliver, Thomson, Murray, Nela and others *(not standardized; no quantitative score; no light standard; poor administration; changes in color; not sensitive for detection of anomalous subjects)*

BEADS—Edridge-Green *(results difficult to interpret; many loose parts; colors not standardized)*

CARDS—Philip *(more useful for detection of color weakness than color blindness; many loose parts; no standard illuminant)*

B. MATCHING TESTS:

Nagel anomaloscope *(Rayleigh Equation—sodium, thallium and lithium lights. Technical difficultics; inconvenient, inaccessible)*

Rand anomaloscope *(in production stage)*

Collins' papers *(variable; Bradley papers; loose parts; no standard illumination)*

ISCC chips—*(time consuming; various modifications under study)*

ISCC aptitude test—*(time consuming; various modifications under study)*

C. NAMING TESTS:

LANTERNS—Edridge-Green lantern, Williams lantern and others *(These are luminous signal tests. They need to be evaluated with reference to reflected signal tests. All luminous signal tests are dependent on source and outside illumination. They utilize small test objects, which yield small retinal images with high retinal brightness.)*

New lanterns are being developed in the Royal Canadian Air Force and the United States military services.

D. DISCRIMINATION AND SENSITIVITY TESTS:

Filters, spectrometers *(laboratory instruments; inaccessible, inconvenient, require standardization)*

Houston filters *(laboratory apparatus, good for special work)*

Nagel cards *(use of color names required; no standard illuminant; time consuming)*

Pseudoisochromatic plates

Stilling *(illuminant not standardized; complicated instructions; no exact diagnosis)*

Ishihara *(good rough screening test; no exact diagnosis; score not indicative of degree of defect)*

Rabkin *(with proper illuminant and administration, good practical test for screening and classification)*

American Optical Company selection *(poor selection of plates; reproductions frequently inaccurate; no critical score)*

Hardy-Rand-Rittler *(illumination and colors standardized; test planned for screening plus qualitative plus quantitative diagnosis; in production)*

E. HUE AND CHROMA SERIES ARRANGEMENT:

NIIP Discs *(used in England for color aptitude testing)*

Murray's Cards *(promising test, in research stage)*

Farnsworth-Munsell 100-Hue Test *(excellent test; many loose parts; complicated scoring; qualitative but not quantitative classification)*

Farnsworth Dichotomous Test *(excellent for qualitative classification of extreme defects; loose parts; does not screen persons with weak defective color vision from those with normal color vision)*

V. A CLASSIFICATION OF TYPES OF COLOR VISION

NORMAL COLOR PERCEPTION

1. NORMAL TRICHROMASY *(three primaries; normal mixtures. Discriminates: light-dark, yellow-blue, red-green)*

h—high or excellent color preception

m—medium or average color preception

l—low or poor color perception, not sufficient to be called defective. Approaches low discrimination type of defect

DEFECTIVE COLOR PERCEPTION

1. LOW DISCRIMINATION *(depression of all three primaries sufficient to amount to a defect)*

x—mild (approaches low normal)

y—medium

z—marked (approaches monochromasy)

2. ANOMALOUS TRICHROMASY *(three primaries; anomalous mixtures)*

(a) Protanomalous (defective red sensitivity; narrowed luminosity curve. Discriminates: light-dark; yellow-blue; red-green poorly)

x—mild

y—medium

z—marked (approaches dichromasy, protanopia)

(b) Deuteranomalous (defective green sensitivity; normal luminosity curve. Discriminates: light-dark; yellow-blue; red-green poorly)

x—mild

y—medium

z—marked (approaches dichromasy, deuteranopia)

(c) Tritanomalous (defective violet sensitivity; normal luminosity curves [?] Discriminates: light-dark; red-green; yellow-blue poorly)

3. **DICHROMASY** *(two primaries)*

(a) Protanopia (first process gone; formerly called "red blindness"; shifted luminosity curve; neutral points at 493 millimicrons and 493 c)

p—pure type (discriminates: light-dark, yellow-blue)

q—complicated type (discriminates: light-dark; yellow-blue poorly)

(b) Deuteranopia (second process gone; formerly called "green blindness"; no shift in luminosity curve; neutral points at 497 millimicrons and 497 c)

p—pure type (discriminates: light-dark; yellow-blue)

q—complicated type (discriminates: light-dark; yellow-blue poorly)

(c) Tritanopia (third process gone; formerly called "violet blindness"; no shift in luminosity curve [?]; neutral points at 572 millimicrons and 572 c)

p—pure type (discriminates: light-dark; red-green)

q—complicated type (discriminates: light-dark; red-green poorly)

4. **MONOCHROMASY** *(one primary; no hue discrimination; "total color blindness—achromatopsia"; discriminates light-dark).*

23 East Seventy-Ninth Street.

TRANSPLANTATION OF HUMAN VITREOUS

A Preliminary Report

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I HAVE not been able to discover any references in the literature to the transplantation of human vitreous from the eye of one person to the eye of another person. It is believed that the cases reported here are the first recorded instances of this procedure. This report is based on some as yet unpublished experiments on transplantation of vitreous in animal eyes.

HISTORICAL REVIEW

Although there are no reported cases of the transplantation of human vitreous, there have been various attempts to provide a clearer vitreous by other methods.

In 1890 Ford [1] withdrew vitreous from several eyes, the loss being made up by the aqueous. He reported improvement in visual acuity from perception of light to 20/60 in 1 case. Deutschmann,[2] in 1906, reported on the injection of vitreous from calf and rabbit eyes into human eyes for the treatment of detachment of the retina. Of 67 patients, the condition of 26 was purportedly improved and that of 38 was unimproved. Komoto,[3] in 1910, reported on the withdrawal of vitreous and injection of saline solution, and Elschnig,[4] on the injection of air. The results of similar procedures were reported by a number of authors between 1920 and 1930, notably by zur Nedden [5] in 300 cases of withdrawal of vitreous.

It was decided to transplant clear vitreous into the eyes of patients with persistent and unabsorbed hemorrhage in the vitreous who had normal intraocular tension and good light projection. Two of the patients had had injuries to the eyes, and 1 had apparently had a spontaneous hemorrhage.

1. Ford, V.: Proposed Surgical Treatment of the Opaque Vitreous, Lancet **1**:462, 1890.

2. Deutschmann, R.: Zur operativen Behandlung der Netzhautablösung, Klin. Monatsbl. f. Augenh. **4**:364, 1906.

3. Komoto: Ueber Glaskorperwaschung bei unheilbarer Glaskorperblutung, abstracted, Klin. Monatsbl. f. Augenh. **50**:265, 1912.

4. Elschnig, A.: Ueber Glaskorperersatz, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch., 1911, p. 514.

5. zur Nedden: The Curative Value of Aspiration of the Vitreous, Arch. Ophth. **57**:109 (March) 1928.

PROCEDURE

With the patient under sodium pentothal anesthesia, the sclera is exposed in the selected quadrant through a conjunctival incision, and a small incision is made with a cataract knife in the equatorial region between a previously placed 0000 silk mattress suture. An 18 gage needle on a 5 cc. syringe is introduced into the vitreous chamber under direct observation by the assistant. Approximately 1.5 cc. of vitreous is withdrawn and the syringe then detached. A syringe containing approximately 2 cc. of clear vitreous from an eye enucleated a few minutes before and kept in an incubator at 37 C. is then attached to the needle. While the assistant exerts tension on the partially tied suture, the vitreous is injected until the eye has reformed. The needle is quickly withdrawn and the suture pulled up, and the tie is completed. Atropine sulfate, 1 per cent, is instilled and a binocular dressing applied.

POSTOPERATIVE TREATMENT

Mild sedatives are used as indicated for two nights, and codeine and acetylsalicylic acid suffice for control of pain. The patient is kept on his back with a pillow for two days, after which he is allowed to turn and be elevated. He is permitted to be up on the fourth or fifth day. There has been no postoperative discomfort. Two patients had a ring of diathermy punctures made at the operative site approximately two weeks prior to transplantation with the idea of stimulating the formations of scar tissue to prevent detachment of the retina. Although this has not been done in all cases, it is believed to be a worth while precaution.

The reports of 3 cases of transplantation of the vitreous follow.

REPORT OF CASES

CASE 1.—*Recipient I. V. H.: Negro; blood group O.*

Note on Admission: The patient gave a history of "spots" before the left eye with recurrent visual loss at various times for two years. On June 9, 1944, while taking physical training, he noticed sudden loss of vision in the left eye. Visual acuity in this eye was reduced to perception of light. He was treated at a station hospital from June 9 to July 19, when he was transferred to the Army general hospital with which I am associated.

Examination: Right eye: The eye was normal throughout. Vision was 20/20.

Left eye: The external condition was normal. Ophthalmoscopic examination revealed a black reflex; no details of the fundus were visible. Examination with the slit lamp showed that the anterior chamber and the lens were normal; the vitreous was filled with a great many golden brown particles, suggestive of hemorrhage. Vision was limited to perception of light; projection was excellent.

Diagnosis: A diagnosis of spontaneous hemorrhage in the vitreous was made. A complete survey revealed nothing of significance except for syphilis, for which the patient was under treatment. He was returned to duty Sept. 9, 1944.

On Jan. 6, 1945 the patient was readmitted to the hospital. He had not noticed any change in vision since his discharge. Vision was 20/20 in the right eye and was limited to perception of light, with good projection, in the left eye. The tension was 18 mm. in each eye.

Donor J. O. F.: Age 27, white; blood group A.

Note on Admission: The patient was wounded in action June 30, 1944 by shell fire. He was admitted to this Army hospital Nov. 2, 1944. He gave no previous history of ocular trouble.

Examination: Right Eye: There was dense corneal opacity with a staphyloma over the lower three fifths of the cornea. Tension was 3 +. Vision was limited to light perception, with faulty projection.

Left eye: The eye was normal. Vision was 20/30 without correction.

Diagnosis: The diagnosis was secondary glaucoma of the right eye. Enucleation was advised because of poor light projection and the unsightly eye.

Operation.—On Feb. 19, 1945, with the patient under sodium pentothal anesthesia, the left eye was enucleated, and a basket type implant used. The eye was placed in isotonic solution of sodium chloride at a temperature of 37 C. On an adjoining table, the left eye of patient I. V. H. was prepared and the lower temporal quadrant of the sclera exposed. According to the previously described procedure, 2 cc. of vitreous was then withdrawn from the donor eye through an 18 gage needle into a 5 cc. syringe. This was placed in an incubator at body temperature. Through an equatorial incision in the recipient eye, 1.5 cc. of straw-colored vitreous was withdrawn, using a similar needle and technic. The syringe was removed from the needle and replaced with the syringe containing donor vitreous, which was slowly injected. The globe, which had become folded in with the withdrawal of vitreous, now reformed. The needle was withdrawn while the assistant pulled up the suture, which was then tied and cut. The conjunctiva was closed with 0000 interrupted surgical gut. Atropine sulfate, 1 per cent, was instilled and a binocular dressing applied.

Postoperative Course.—The eye was redressed on the third postoperative day, and 2 per cent atropine sulfate was instilled. There was little reaction.

February 25: No unusual reaction followed transplantation of the vitreous. The vitreous showed considerable clearing. The disk was seen indistinctly, and a number of the diathermy punctures were also visible. Vision had improved to perception of hand movements at 2 feet (60 cm.).

February 27: The details of the fundus could be made out in the section of the periphery extending approximately from 9 to 3 o'clock. On the temporal side there was a small retinal hemorrhage at about 2:30 o'clock. The disk could not be seen, although it was visible two days after the operation. Tension was 19 mm. in the right eye and 9 mm. in the left eye.

March 1: The disk could be distinguished clearly, as well as the arteries and veins in the midperipheral area below and in the region of the macula. The extreme periphery above and temporally could be seen, as noted in the previous examination. There was a very slight reaction remaining.

March 2: Tension was 15 mm. in the right eye and 11 mm. in the left eye. The fundus was unchanged.

March 7: Tension was 18 mm. in the right eye and 13 mm. in the left eye. The posterior pole of the fundus remained visible. Apparently, the fresh vitreous had diluted some of the old, opaque vitreous, to give a somewhat hazy view of the posterior pole. The macular area was visible as a dark reddish spot, with the appearance of an old degeneration.

March 21: Tension was 19 mm. in the right eye and 14 mm. in the left eye. The eye was quiet. The vitreous remained moderately clear.

April 18: Tension was 17 mm. in the right eye and 16 mm. in the left eye. Vision in the left eye was 20/200; with the pinhole disk it was 20/70 +, and the patient read Jaeger test type 10.

May 3: The media remained clear. The disk and the areas previously noted were well seen, as was the dark area in the macula. Vision in the left eye

was 20/100; with—1.00 D. cyl., axis 180 it was 20/60—1. Tension was 17 mm. in each eye.

The patient was discharged to duty June 6, 1945.

The patient was seen in the clinic on September 28. At this time the tension was 17 mm. in each eye. The left eye was white and quiet. Ophthalmoscopic examination of this eye showed some floaters, but no more than on previous examinations. The disk, the vessels and the macular area were distinct. The periphery was clearly seen except below, where some remnants of old hemorrhage were present in the vitreous. Vision was 20/100 in each eye; with —1.00 D. cyl., axis 180 it was 20/60—1.

Comment.—No unusual reaction was evident postoperatively. The eye showed only a slight conjunctival injection, equivalent to what is sometimes seen after discission of a secondary membrane. Examination with the slit lamp showed no evidence of iritis. The tension became normal approximately one month later. The vitreous showed considerable clearing during the first few days, and the area of clear vitreous became greater during the first month. Vision became stabilized at the end of two months. The improvement was still present seven months after operation. From the appearance of the vitreous it was felt that vision would be considerably better if it were not for the macular change.

CASE 2.—*Recipient A. F. H.: Age 23, white; blood group O.*

Note on Admission: The patient was involved in a fist fight on Dec. 25, 1944, when he was struck in the left eye and lost consciousness. Examination at an overseas hospital on December 29 revealed that vision was 20/20 in the right eye and limited to light perception in the left eye, with proptosis and hemorrhage into the anterior chamber. The tension was elevated. While he was under treatment, the hemorrhage of the anterior chamber absorbed, revealing a remaining hemorrhage in the vitreous. The patient was examined at this Army hospital on Feb. 17, 1945.

Examination: Right eye: The eye was normal throughout.

Left eye: The external condition was normal. The fundus gave a black reflex. Examination with the slit lamp revealed a large amount of mobile blood pigment in the vitreous. Vision was 20/20 in the right eye and was limited to light perception in the left eye. Tension was 15 mm. in the right eye and 16 mm. in the left eye. Roentgenographic examination revealed no foreign body.

Diagnosis: The diagnosis was post-traumatic hemorrhage of the vitreous.

Course: Examination on March 8 revealed a small amount of red blood in the extreme lower part of the fundus.

Donor W. E. A.: Age 25, white; blood group O.

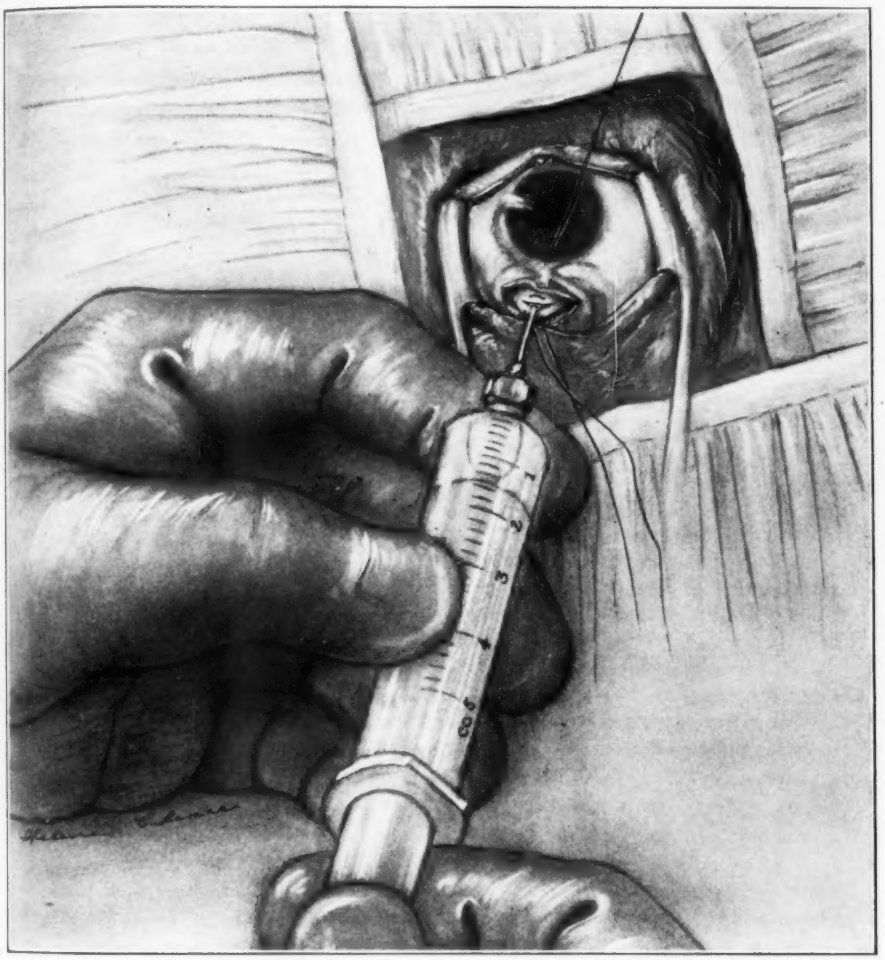
Note on Admission: The patient had been hit in the left eye with BB shot at the age of 9 years, after which he had some type of operation and later lost the sight of the eye.

Examination: Right eye: The eye was normal throughout.

Left eye: The cornea was thick and somewhat opaque. The anterior chamber was very shallow, with a dense pupillary membrane. Tactile tension was normal. Vision was 20/20 in the right eye and was limited to light perception, with faulty projection, in the left eye.

Diagnosis: The diagnosis was occlusion of the pupil and possible detachment of the retina in the left eye.

Operation.—On March 9, 1945 a transfusion of 2 cc. of vitreous was attempted. However, no vitreous could be withdrawn through a 15 gage needle. On withdrawal of the needle some clear vitreous escaped, and approximately 1.5 cc. of vitreous was injected into the recipient eye from the donor eye of W. E. A., enucleated prior to the transplantation of vitreous.



Withdrawal of cloudy vitreous (1.5 cc.).

Postoperative Course.—March 12: A small amount of fresh hemorrhage was noted on the lower pupillary border. Tactile tension was normal. There was moderate injection of the bulbar conjunctiva. There was also evidence of additional fresh hemorrhage in the anterior vitreous, below. A vessel could be seen on the temporal side, suggestive of detachment of the retina.

March 21: Tension was 17 mm. in the right eye and 20 mm. in the left eye. The vitreous still had a large amount of mobile blood. Vision was limited to perception of hand movements at 6 feet (180 cm.). Projection was poor on the temporal side.

The patient was discharged from the hospital on Aug. 26, 1945.

Comment.—There was the impression at the operation that the needle did not penetrate into the vitreous chamber, but remained behind the retina, and that the patient probably had a retinal detachment. This impression was strengthened by the loss of clear, fluid vitreous, rather than cloudy vitreous, when the needle was withdrawn. The fact that the patient had further hemorrhage after operation suggested that the vascular structure of the eye was not in a healthy condition. The injection of vitreous probably increased the detachment. At the time of the patient's discharge the eye was entirely quiet. There was no evidence of fresh hemorrhage. The tension was normal. Unfortunately, the light projection, which was not good after operation, had not been recorded before operation. The procedure, however, was a complete failure.

CASE 3.—*Recipient W. S.: Age 27, white; blood group A.*

Note on Admission: The patient stepped on a land mine in France on Aug. 29, 1944 and was injured in both eyes, the legs, the right arm and the chest. The right eye was enucleated shortly after injury. It was noted at an overseas hospital, on September 2, that there was some red blood in the vitreous chamber of the left eye. The patient was examined at this Army hospital on Nov. 17, 1944.

Examination: The right eye had been enucleated.

Left eye: Vision was 20/300. Small maculas and foreign bodies lay on the surface of the cornea. A corneal scar was present at 2 o'clock, with some embedded debris. A complete iridectomy of the upper temporal quadrant had been performed. The anterior chamber was of normal depth. The pattern of the iris was well preserved. The pupil reacted to light. There was a slight capsular opacity of the lens posteriorly. Ophthalmoscopic examination revealed a dense vitreous opacity occupying all but the extreme upper periphery, where the vessels could be hazily seen.

Diagnosis: The diagnosis was hemorrhage of the vitreous, and transplantation of vitreous was suggested.

Donor B. D. N.: Age 28, white; blood group O.

Note on Admission: The patient was struck in the left eye on Nov. 8, 1944, resulting in a penetrating wound of the left globe. Vision was limited to perception of hand movements. He was seen in this Army hospital on December 13.

Examination: Right eye: The eye was entirely normal.

Left eye: Inspection showed a healed laceration of the lids with slight ptosis. There was a dense leukoma of the cornea, with a disorganized iris adhering to it. The lens was cataractous. The fundus could not be seen.

Vision was 20/50 in the right eye (20/15 with correction) and was limited to perception of hand movements at 1 foot (30.4 cm.), with poor projection, in the left eye.

Diagnosis: The diagnosis was occluded pupil, cataractous lens and possible retinal detachment.

Operation.—On April 12, 1945 vitreous was withdrawn from the recipient eye through a temporal incision at the site of the diathermy punctures, and approximately 2 cc. of vitreous was injected from the donor eye of B. D. N.

Postoperative Course.—April 18: The slight ciliary flush, which had been present for three days, had practically disappeared. A good red reflex was visible over the upper one half to two thirds of the fundus, and retinal vessels could be clearly seen in the macular area and one half of the disk itself. The tension was low.

April 20: The disk could be seen fairly well, although the media was slightly hazy. There was a large vitreous floater which temporarily obscured vision when the patient moved the eye suddenly.

During the following three weeks the eye became entirely white. The tension gradually improved, and the upper half of the fundus could be seen fairly well.

May 15: Tension was 12 mm.

May 31: Vision was 20/40; with a —1.00 D. sphere it was 20/20—2. There was considerable cloudy vitreous in the lower two fifths of the fundus.

July 13: Vision was 20/40 and was correctible to 20/20. Tension was 13 mm. The details of the fundus in the upper temporal quadrant were visible well out into the periphery, as well as in the macular region. The details could also be seen well down the temporal side to 4 o'clock. On examination with the slit lamp the cornea, the anterior chamber and the iris appeared as they had on admission.

August 4: The fundus was unchanged except for the gradual clearing of the vitreous below on the temporal side. An opacity remained in the lower nasal quadrant of the vitreous. No lesion was evident in the fundus except for the hemorrhage in the vitreous and a choroidal scar, which could be seen in the upper temporal quadrant at the site of injection. Vision was 20/40 in the left eye; with —0.50 D. sph. ⌒ —0.50 D. cyl., axis 155 it was 20/20. Tension was 13 mm.

The patient was discharged from the hospital and from the Army on Aug. 6, 1945.

Comment.—This patient was on the program for the blind and was scheduled to continue his rehabilitation training. However, because of his improved vision, he was discharged directly to his home. The visual field was very good except for the opacity below, which interfered slightly with his vision on sudden movement, to such an extent that it was not possible at the time of his discharge to drive a car. However, he was able to read Jaeger type 1, read the newspapers and carry on his correspondence and had no difficulty in traveling about wherever he wanted to go.

SUMMARY

Approximately ten years ago I conducted some experiments on laboratory animals, which have not yet been published. These experiments demonstrated that the transplantation of vitreous from the eye of one animal to that of another of the same species is a safe and practicable procedure. Publication was delayed until an opportunity should present itself to conduct this procedure on suitable human eyes.

It is well known that the absorption of a hemorrhage in the vitreous may sometimes be long delayed, but after many months it will clear remarkably. In other cases retinitis proliferans with vitreous bands and adhesions will develop, with disastrous results to the eye. Whether transplantation should be tried early or late is difficult to decide. Probably in cases of severe hemorrhage the earlier a vitreous transplantation is done after bleeding stops, the better. It so happened that the only cases available in this Army hospital have been those of old hemorrhage into the vitreous chamber. It is possible that further absorption of these hemorrhages would have taken place if nothing had been done. However, it is a not uncommon experience to see retinitis proliferans develop within a short time.

In all cases in which this procedure was used it was discussed with the patients, and it was explained to them that this was an experimental and untried method so far as human eyes were concerned. They all expressed a desire to have the operation.

In no cases in which the operation has been done has the eye been lost or apparently become worse than it was before operation. Further investigation is under way, but much still remains to be done. This applies to the actual operative procedures as well as to the type of cases selected. In a case of hemorrhage in the vitreous one is working very much in the dark, and further knowledge is necessary in order that one may be able to determine which cases are suitable and when operation should be performed. This implies a study and correlation of the clinical appearance and the pathologic changes together with chemical studies on the abnormal vitreous. The possibilities of this procedure in treatment of other conditions, such as detachment of the retina, are still to be investigated. Repeated transplantations of vitreous have not yet been done.

One cannot, of course, introduce instruments into the vitreous chamber with impunity, but it is felt that the procedure described here should open up a new field for surgery in selected cases in which the treatment until now has been, to say the least, expectant.

The observations on the 3 cases reported may be summarized as follows:

In 2 of 3 cases in which transplantation of vitreous was done, the procedure was successful up to the time of this report.

From 1.5 to 2 cc. is the maximum amount of vitreous which can be withdrawn through a 15 or 18 gage needle from the recipient eye.

The blood group does not appear to be significant.

There was a decided reduction in the opacity of the vitreous in the cases in which the operation was successful.

The pathologic vitreous appeared to be more fluid than normal, but it was not possible to determine whether it became more fluid with the admixture of transplanted vitreous.

There was a moderate reaction after the procedure, but no evidence of a foreign protein reaction was noted.

The tension in all 3 cases returned to normal within a reasonable period and remained normal.

The procedure was successful in cases of spontaneous as well as of post-traumatic hemorrhages in the vitreous.

SUPRASELLAR MENINGIOMAS ASSOCIATED WITH SCOTOMATOUS FIELD DEFECTS

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AND

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PHILADELPHIA

SUPRASELLAR meningiomas are associated in typical instances with atrophy of one or both optic nerves and bitemporal hemianopsia, with or without changes in the sella turcica and with or without symptoms of endocrine disturbance. In some circumstances, optic nerve atrophy may be minimal or altogether absent, the only evidence of chiasmal compression being a bitemporal hemianopsia for form or color or both. In almost all instances, the symptoms are slowly progressive, with visual loss prominent and headache equally disabling.

Not sufficiently well recognized as part of the suprasellar tumor syndrome is the occurrence of sudden visual loss with scotomas in the visual fields, associated with more or less prominent headache. The development of symptoms may be so rapid and may simulate so closely in the early stages the clinical picture of retrobulbar neuritis that great care must be exercised to avoid discarding a diagnosis of suprasellar meningioma. For this reason, we believe it is desirable to report our experience with 4 cases which illustrate the points in question.

REPORT OF CASES

CASE 1.—*A woman aged 31 admitted on Sept. 16, 1939, with history of failing vision for eight months, including period of partial remission; bilateral atrophy of optic nerve with central scotoma in left eye and a combined central and peripheral field defect in right eye; progression, with severe headaches for a year prior to death, on April 19, 1942, after operation. Verified diagnosis of suprasellar meningioma.*

History.—E. H., a woman aged 31, was admitted to the Wills Hospital on Sept. 16, 1939, to the service of Dr. Louis Lehrfeld. The family history was noncontributory. The past medical history revealed that the patient gained weight excessively during her first pregnancy, which resulted in a miscarriage, and that she was underweight and anemic during her second pregnancy, which terminated, in 1935, in prolonged labor, followed by delayed extraction of the placenta. Some irregularity in menstruation and a sense of numbness in the arms and legs were experienced during 1938; an abscessed tooth was treated in January 1939.

From the Department of Neurology, Jefferson Medical College of Philadelphia.

The present illness developed suddenly in February 1939, when the patient, after looking out of a window at ground covered with snow, noticed impairment of vision. She became panicky and depressed. The visual impairment became progressively worse, so that a month later she could only see objects within several feet of her. Treatment, including medication in the form of parenteral injections of thiamine hydrochloride, was followed by gradual improvement in vision, so that by June 1939 visual acuity was 20/100 in the left eye and 20/50 in the right eye. This condition continued for about one month and then gradually regressed. The patient became increasingly apprehensive and also manifested emotional instability in the form of crying. She complained of "flakes of light" in both eyes.

Examination.—On admission the patient appeared rather poorly nourished; her heart, lungs and abdomen were grossly normal.

Neurologic examination revealed the station to be within normal limits. The ordinary gait was normal, but there was unsteadiness in the heel to toe test. There was no evidence of incoordination in any of the extremities. Motor power was well preserved throughout. The tendon reflexes were equally active in the arms

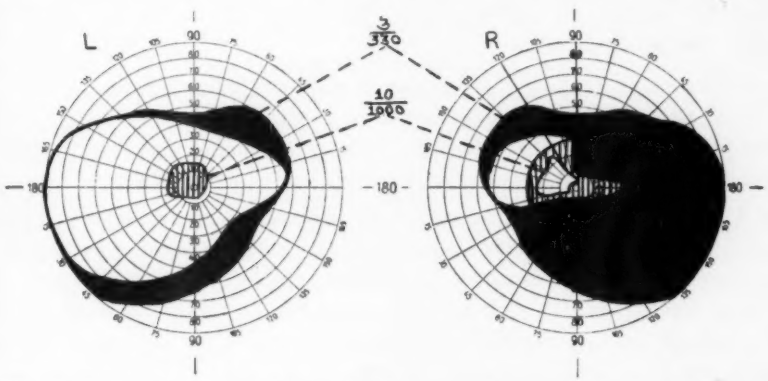

Fig. 1 (case 1).—Visual fields, showing central scotoma in the left eye and temporal hemianopsia with nasal pericentral ring scotoma in the right eye.

and legs. The abdominal reflexes were equally active on the two sides. No pathologic reflexes were noted. Pain, position and vibration sensations were intact throughout.

The fundi showed pallor of both optic disks. Visual acuity was 6/30 in the right eye and 1/60 in the left eye. There were a central scotoma in the left eye and a combined central and peripheral visual field defect in the right eye (fig. 1). The pupils were equal and regular and reacted well to light and in accommodation. The extraocular movements were normal. The corneal reflexes were equally active. No facial, palatal or lingual weaknesses were observed.

Laboratory Data.—A blood count revealed 78 per cent hemoglobin, 4,110,000 red cells, 7,900 white cells and a color index of 0.95 with 76 per cent neutrophils 22 per cent lymphocytes and 2 per cent monocytes. The urine was normal. The serologic reactions of the blood were negative for evidence of syphilis. The spinal fluid was clear and colorless and was under an initial pressure of 190 mm.; it contained 1 cell per cubic millimeter; the serologic reactions were negative

and the colloidal gold curve was normal. A roentgenogram of the skull showed slight demineralization of the dorsum sellae but was otherwise normal.

Course.—After a short period of observation, the patient was discharged from the hospital unimproved. Visual impairment progressed gradually, and two years later, in 1941, severe headaches appeared. The headaches recurred at increasingly frequent intervals and were unrelieved by acetylsalicylic acid. Six months later the headaches were associated with projectile vomiting. In January 1942, three years after the onset of symptoms, a mental disorder became evident in conjunction with the development of an abscess of the breast. On admission to the Germantown Hospital, on Feb. 9, 1942, the patient was found to be exhausted and moderately anemic. She was somnolent and emotionally labile. The significant neurologic observations were amaurosis, a dirty gray pallor of the optic disks and dilated pupils, which did not respond to light but reacted in accommodation. An electroencephalogram showed abnormal slow waves throughout both hemispheres. A roentgenogram of the skull was normal. A ventriculogram showed changes indicative of a suprasellar neoplasm. Craniotomy revealed a suprasellar meningioma, originating in front of the optic chiasm but extending back over the sphenoid ridge into the right middle fossa. Three days after a second craniotomy, for the purpose of completing removal of the tumor, the patient died, on April 19.

Comment.—This case illustrates clearly the problems under discussion. The onset of the visual loss was sudden and was so rapidly progressive that almost all vision was lost in the course of a month. The optic disks were pale at the time of the patient's first admission, and the visual fields revealed scotomatous defects. The subsequent course was characterized by further loss of vision, the development of headache and more pronounced atrophy of the optic nerve.

The symptoms at the time of onset were much more typical of retrobulbar neuritis than of a suprasellar tumor, as evidenced by the sudden loss of visual acuity and the scotomas. The occurrence of partial remission of impaired vision in this case following intensive vitamin therapy was another feature which could easily be misinterpreted as favoring the diagnosis of neuritis. Only with the appearance of persistent headache was the diagnosis of tumor seriously regarded and eventually established.

CASE 2.—*A girl aged 16, admitted on Jan. 18, 1940, with history of headaches for three years and impairment of vision for four months; bilateral atrophy of optic nerve, with combined central and peripheral field defects in both eyes; death on Feb. 10, 1940, following operation; verified diagnosis of suprasella meningioma.*

History.—B.D., a girl aged 16 years, was admitted to the neurologic service of the Jefferson Medical College Hospital on Jan. 18, 1940. The family history was noncontributory. The past medical history revealed that the patient had been in good health except for childhood diseases.

The onset of the present illness occurred three years previously, with moderately intense headaches in the occipital region, lasting one or two hours and recurring at weekly intervals. At that time, the patient had impairment of distant vision, but near vision was good. After refraction vision was completely satisfactory.

The headaches also became less frequent and less intense but continued to recur at intervals of one or two months. Episodes of vertigo first developed in September 1939, at which time it was noticed that the patient was holding objects closer to her eyes while reading. About one month later the patient awoke during the night with vomiting and an excruciating headache, which persisted for one or two days. A similar episode of headache and vomiting occurred two weeks later. Headaches and vertigo continued to recur frequently thereafter. One month after this she showed evidence of impairment of memory, which was noticed by members of her family, and still another month later gradually increasing impairment of vision developed. Her condition remained unchanged until Jan. 2, 1940, when she awoke in the morning to discover that her vision had failed to such a degree that she was unable to read. Several hours later a severe occipital headache developed, persisted for a short time and recurred at irregular intervals throughout the day. During the next day, vision remained blurred and the headache returned. On one occasion that day, when she was assuming the erect position, vertigo appeared and persisted for five minutes. The headaches continued to recur daily, and the episodes of vertigo reappeared occasionally. At times the patient noticed an aching sensation in her eyes. Impairment of memory and concentration were marked a few days before her admission to the hospital.

Examination.—Physical examination revealed fairly good development. The thyroid was slightly enlarged. The nose, throat and sinuses showed no evidence of infection. The lungs and abdomen were grossly normal. The heart was slightly enlarged, and there was a presystolic murmur over the apex. The blood pressure was 114/82.

Neurologic examination revealed unsteadiness in standing on either leg alone and in the heel to toe test. There was no evidence of incoordination in the finger to nose or the heel to knee test. Motor power was well preserved throughout. There was generalized tendon hyporeflexia. The abdominal reflexes were diminished and exhaustible. No pathologic plantar reflexes were noted. Pain, position and vibration senses were intact throughout.

The sense of smell was well preserved. The right fundus showed slight temporal pallor of the optic disk, with the lamina cribrosa visible in a broad, deep cup. The left fundus showed pronounced silvery gray pallor of the entire optic disk with slight edema of the nasal margin, and the lamina cribrosa was not visible in the temporal portion of the cup. There was some fulness of the retinal veins bilaterally; the retinal arteries appeared normal. Visual acuity was 5/200 in each eye. Examination of the visual fields showed peripheral and central defects in both eyes (fig. 2). The pupils were large, equal and regular and reacted well to light and in accommodation. Extraocular movements were normal. No facial, palatal or lingual weaknesses were observed.

Laboratory Data.—The urine was normal. The blood count showed 72 per cent hemoglobin, and 4,000,000 red cells, 8,500 white cells, a color index of 0.88, with 79 per cent neutrophils, 20 per cent lymphocytes and 1 per cent monocytes. The serologic reactions of the blood were negative for evidence of syphilis. The blood contained 29 mg. of nonprotein nitrogen, 12.34 mg. of urea nitrogen and 90 mg. of sugar, per hundred cubic centimeters. Urea clearance was 100 per cent, average normal volume of urine per minute. The spinal fluid was clear and colorless; the initial pressure was 150 mm.; the fluid contained less than 1 cell per cubic millimeter, the Wassermann reaction was negative, and the total protein measured 54 mg. per hundred cubic centimeters. The electrocardiogram showed

a left axis deviation but was otherwise normal. The Bárány test revealed abnormal reactions, which were interpreted as being the result of "a lesion in the region of the decussation of the superior cerebellar peduncles." A roentgenogram of the skull was normal. A roentgenogram of the sinuses disclosed evidence of chronic hyperplastic ethmoiditis bilaterally and right frontal sinusitis.

Course.—After her admission to the hospital, the patient did not complain of headaches, but the pronounced impairment of vision persisted. Repeated

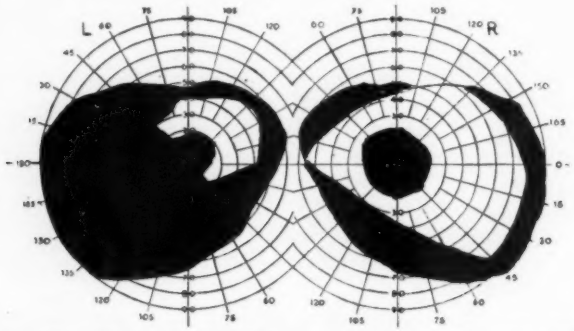

Fig. 2 (case 2).—Visual fields (10 mm. test object; distance, 330 mm.; daylight), showing central scotoma merging inferiorly with temporal hemianopsia in the left eye and central scotoma in the right eye. Vision was limited to counting fingers at 6 inches (15 cm.) in each eye (eccentric).

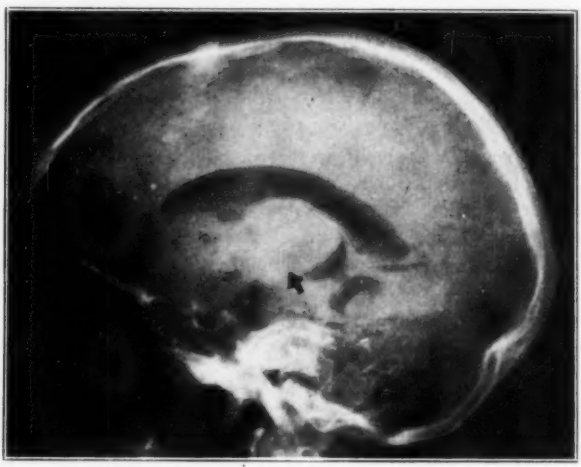

Fig. 3 (case 2).—Pneumoencephalogram, showing obliteration of cisterna chiasmatis and encroachment on the floor of the third ventricle.

examination of the fundi during the first week after her admission disclosed slightly increasing edema of the nasal margin of the left optic disk, with extension to the upper and lower margins, but without measurable elevation. The appearance of the fundi was unchanged during the second week.

Pneumoencephalographic examination (fig. 3) was performed, and the report follows: "Obliteration of the cisterna chiasmatis was seen in the lateral view.

In this region there was a sharply demarcated shadow, suggestive of a space-taking lesion in the suprasellar region. The third ventricle was encroached on, and there may have been some elevation of the right lateral ventricle, as seen in the sagittal view."

Reexamination of the visual fields one week after this showed enlargement and alterations in outline of the defects bilaterally (fig. 4).

A right frontal craniotomy was performed on February 7, and the report follows: "The frontal lobe was retracted, and a mulberry-like, pink mass was found near the midline. The right optic nerve was seen to be widely stretched over the neoplasm, which protruded laterally beneath the nerve. The major portion of this mass appeared in the anterior fossa medial to the right optic nerve, although it extended caudal to the medial end of the right sphenoid ridge."

After operation, the patient remained comatose, with progressive elevation of temperature to a level of 108 F. at the time of her death, on February 10.

Autopsy.—A moderate extravasation of coagulated blood was observed beneath the bone flap in the right frontal and parietal regions. The tip of the right

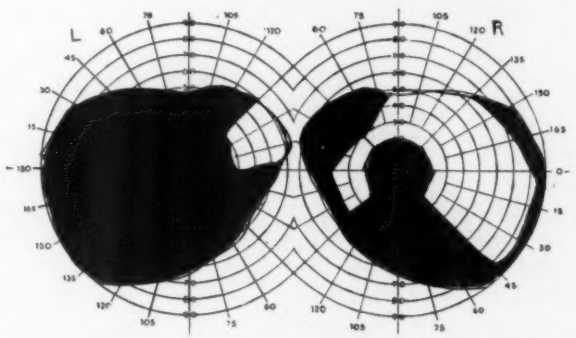

Fig. 4 (case 2).—Fields (10 mm. test object; distance, 330 mm.; daylight) taken two weeks after those shown in figure 2, showing expansion of defect, with vision limited to the superior nasal quadrant in the left eye and merging of central scotoma inferiorly with peripheral indentation in the right eye.

frontal lobe was slightly macerated, and the brain generally was edematous. The area of the chiasmatic cistern was occupied by a firm, fleshy, walnut-shaped tumor (fig. 5), which measured 2.5 by 3.5 by 3 cm. The posterior limit of the tumor was in the optic chiasm, and its anterior limit extended just proximal to an imaginary line joining the tips of the temporal poles. The surface of the tumor had been macerated (after operation). The olfactory bulbs and tracts could not be identified. The interpeduncular fossa was obliterated with hemorrhage. Microscopic examination revealed a meningioma.

Comment.—The problem in this case was somewhat simpler in some respects than that in the first case, but the diagnosis of a suprasellar tumor was still difficult in the absence of typical hemianoptic field defects. The early appearance of headache and its persistence aroused suspicions that a cerebral tumor was present, and the occurrence of atrophy of the optic nerve appeared to confirm the suspicion; but the

presence of scotomatous field deformities was unusual. In this instance, the problem was not complicated by sudden visual loss, but visual acuity was low (5/200 in each eye) and seemed to be far below the acuity to be expected from the appearance of the fundi. On the whole, the possibility of tumor was so definite, largely because of the headache, and despite the features of retrobulbar neuritis, that a pneumoencephalographic examination was performed, revealing a suprasellar tumor. Another noteworthy feature in this case is the age of 16 years, which is consider-

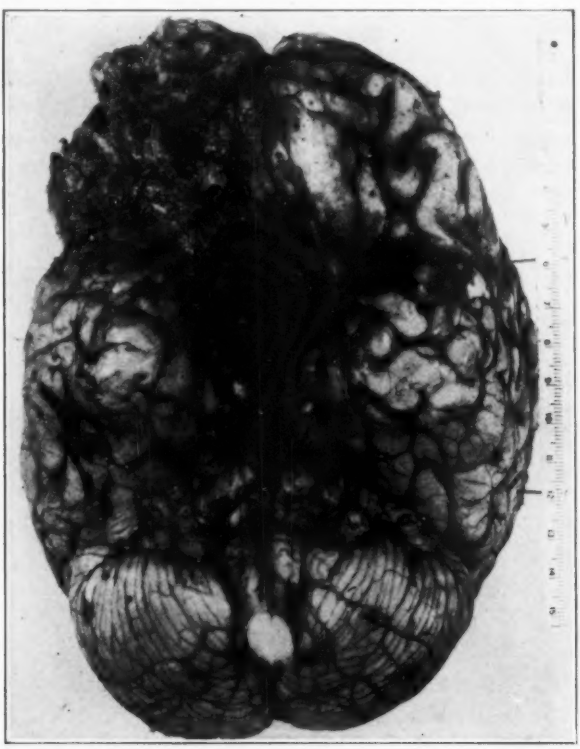

Fig. 5 (case 2).—Basilar view of brain, with suprasellar meningioma occupying cisterna chiasmatis.

ably below the youngest age recorded previously, that of 27 years, reported by Cushing and Eisenhardt [1] in their series of cases of suprasellar meningioma.

CASE 3.—*A woman aged 31, admitted on April 24, 1940, with history of rapidly failing vision of right eye for four months and of left eye for one month; anosmia on left side, bilateral optic nerve atrophy, with papilledema in right eye and*

1. Cushing, H., and Eisenhardt, L.: Meningiomas, Springfield, Ill., Charles C Thomas, Publisher, 1938.

combined central and peripheral field defects of both eyes; progression of visual impairment prior to operation, on May 2, 1940. Patient discharged with verified diagnosis of suprasellar meningioma.

History.—E. H., a woman aged 31, was first observed at the Wills Hospital on the service of Dr. W. J. Harrison and was subsequently admitted to the neurologic service of the Jefferson Medical College Hospital, on April 24, 1940. The family history was noncontributory. The past medical history revealed that the patient had been in good health except for minor diseases of childhood. She was a clerk by occupation and had no history of exposure to any known exogenous poisons.

The onset of the present illness occurred four months prior to her admission to the hospital with blurring of vision and a sensation of mist and a spot before the right eye. Within a short time the patient was unable to read with this eye. At that time she noticed mild frontal headaches, which appeared usually in the morning, but after treatment for supposed sinus infection the headaches completely disappeared. The impaired vision of the right eye became progressively worse. Within the month preceding her admission, impairment of vision of the left eye appeared and progressed rapidly, so that the patient was unable to read with either eye. Double vision was noticed for a short period prior to hospitalization. There was no history of impairment of memory or alteration of personality.

Examination.—Physical examination revealed a relatively slender woman, of small stature. Examination of the nose and throat showed a thickened, hyperplastic mucous membrane in both ethmoid areas. The tonsils were small and embedded. The heart, lungs and abdomen were grossly normal. The blood pressure was 110 systolic and 70 diastolic.

Neurologic examination revealed normal station and gait. There was no evidence of incoordination in any of the extremities. Motor power was well preserved throughout. The tendon reflexes were equally active in the arms. The patellar and achilles reflexes were hyperactive bilaterally, being possibly more pronounced in the right leg than in the left. No pathologic plantar reflexes were noted. Pain, position and vibration senses were intact throughout.

There was anosmia on the left, with odors perceived but not identified on the right. The right fundus showed papilledema of 2 D. at the nasal margin of the optic disk, with the swelling diminishing over the remainder of the disk and a small area free of edema near the inferior temporal margin; in the area which was free of edema a small part of the lamina cribrosa was visible and seemed pale, while the swollen portion of the disk was congested. The left fundus showed definite pallor of the optic disk, most pronounced temporally, with the margins of the disk well outlined and considerable lamina cribrosa visible in the broad, deep cup. The retinal arteries and veins bilaterally were somewhat tortuous, but there was no venous engorgement. The macular area was normal bilaterally. Visual acuity was 1/60 in the right eye and was limited to counting fingers at 18 inches (45 cm.) in the left eye. Examination of the visual fields on April 18, 1940 at the Wills Hospital showed peripheral anl central defects in both eyes (fig. 6). The pupils were dilated and not quite round, with the right pupil reacting sluggishly to light directly and not reacting consensually; the left pupil failed to react to light directly but reacted consensually. There was slight paresis of the external rectus muscle bilaterally, with the extraocular movements otherwise normal. The corneal reflexes were equally active on the two sides. There was no weakness of the face, palate or tongue.

Laboratory Data.—The urine was normal. The blood count showed 75 per cent hemoglobin, 4,500,000 red cells, 4,300 white cells and a color index of 0.83, with 62 per cent neutrophils, 35 per cent lymphocytes and 3 per cent monocytes. The blood contained 29 mg. of nonprotein nitrogen and 114 mg. of sugar, per hundred cubic centimeters. The serologic reactions of the blood were negative for evidence of syphilis. The spinal fluid was blood tinged; the initial pressure was 55 mm.; the Wassermann reaction was negative, and the total protein measured 18 mg. per hundred cubic centimeters. The electrocardiogram was within normal limits.

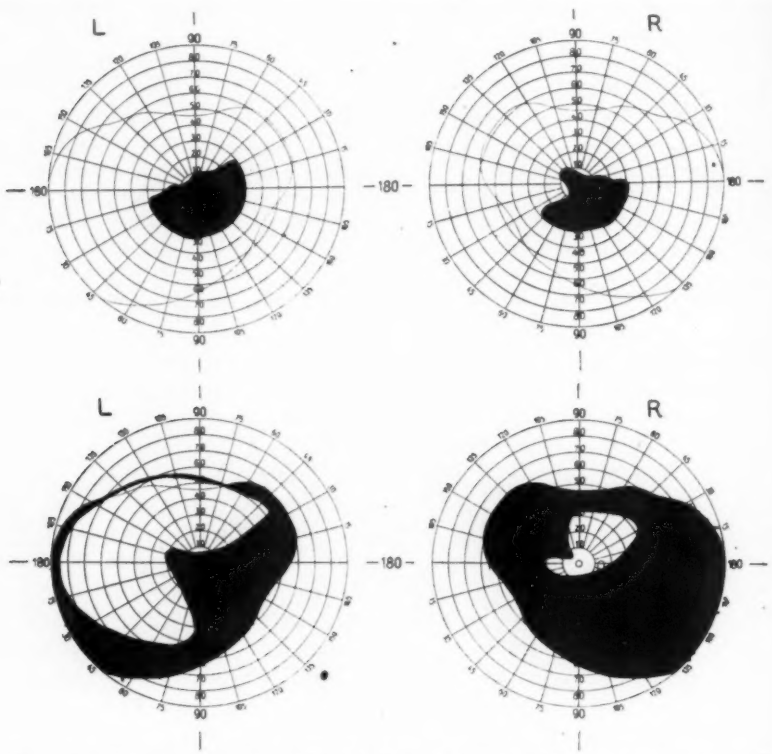

Fig. 6 (case 3).—Visual fields, showing obliquely inferior altitudinal central scotoma bilaterally with peripheral constriction, which is nasal on the left and more uniform on the right.

A roentgenogram of the skull showed haziness of all the sinuses, with evidence of destructive changes involving the anterior portions of the sphenoid bone. The destructive changes were more advanced on the right side. There was no evidence of increased intracranial pressure. The sella turcica was normal in size and shape. The left optic foramen was small and distorted in outline, while the right optic foramen was well visualized.

Course.—Vision continued to become progressively worse after the patient's admission to the hospital. Reexamination of the visual fields eleven days after the original study revealed considerable extension of the field defects (fig. 7).

Craniotomy was performed on May 2, 1940, and the report follows: "After incision of the dura transfrontally and retraction of the right frontal pole, an

encapsulated neoplasm was discovered lying directly in the midline. The neoplasm was at least as large as a lemon and extended caudally as far as could be visualized, while medially it extended beneath the free edge of the falx. The roof of the right orbit appeared distorted and more prominent than normal. After incision of the capsule, tumor tissue, having a honeycombed appearance, was removed and found to be rather vascular. The neoplasm appeared to extend directly into the sphenoid sinus and into the cribriform plate. The optic nerves and anterior cerebral arteries could not be visualized and were believed to be embedded within the tumor. No attempt was made to remove the neoplasm from a wide area." After operation the patient reacted favorably, but vision remained limited to light perception in the right eye at the time of discharge, on May 24, 1940.

Microscopic examination of tumor fragments removed at operation revealed a meningioma.

Comment.—The course of the illness in this instance was rapid, with headaches and development of visual loss over a period of four months. The loss of vision was more prominent than headache and had pro-

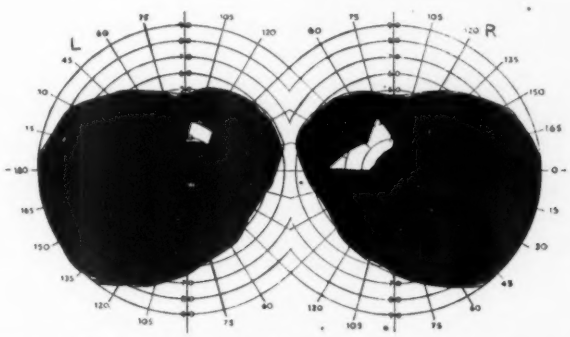

Fig. 7 (case 3).—Fields taken with 15 mm. test object at distance of 330 mm., with daylight illumination, eleven days after those in figure 6, showing expansion of defects bilaterally, with remnant of vision limited to the upper nasal quadrant bilaterally (left eye, perception of hand movements; right eye, counting fingers at 9 inches [22 cm.]).

gressed to such a degree for three months in the right eye and for one month in the left eye that the visual acuity at the time of examination was 1/60 in the right eye and was limited to perception of form at 18 inches (46 cm.) in the left eye. Despite rapid visual loss and presence of scotomas, the possibility of tumor was regarded as good, though it is doubtful whether the diagnosis could have been established with certainty without pneumoencephalographic studies had it not been for the destructive changes in the region of the sella turcica. It was this lesion which established the diagnosis definitely in the present case. It is interesting to note that the headache disappeared completely with treatment of the sinuses. It is also noteworthy that the penultimate stage of visual impairment was reached in the superior nasal quadrants.

This agrees with the observations of Traquair [2] and de Schweinitz,[3] who described the penultimate stage as usually consisting of vision limited to the superior nasal quadrants. On the other hand, Cushing and Walker [4] described the penultimate stage as usually consisting of vision limited to the inferior nasal quadrants.

CASE 4.—*A woman aged 23, admitted Aug. 19, 1942, with history of headaches and increasing visual impairment of left eye for three months, followed by failing vision of right eye for one month; optic nerve atrophy in the left eye with merged central and inferior altitudinal defects in left eye and a central scotoma for color in right eye; progression of visual impairment. Verified diagnosis of suprasellar meningioma.*

History.—C. V., a woman aged 23, was admitted to the Jefferson Medical College Hospital on Aug. 19, 1942. The family history was noncontributory. The past medical history disclosed that the patient had been in good health except for minor diseases of childhood.

The onset of the present illness occurred three months prior to her admission with visual impairment of the left eye associated with dull pain in the left supraorbital region. There were also frequent headaches in the frontal region and occasional headaches in the left temporal area. The pain in the left supraorbital region subsided within three weeks, but the more diffuse headaches persisted. Impairment of vision in the left eye became progressively worse, and within the month before hospitalization gradually increasing visual impairment of the right eye was also noticed.

Physical Examination.—The patient was moderately obese; her heart, lungs and abdomen were grossly normal. The blood pressure was 118 systolic and 70 diastolic. Survey of the nose and throat showed a slightly inflamed and thickened nasal mucosa but no definite evidence of sinusitis.

Neurologic examination revealed no disturbance of station or gait. There was no evidence of ataxia or dyssynergia. The motor power was well preserved throughout. The tendon reflexes were equally active in the arms and legs on the two sides. No pathologic reflexes were elicited. Pain, position and vibration senses were unimpaired.

The fundi showed slight pallor of the left optic disk, more evident on the nasal side. Examination of the visual fields showed an inferior altitudinal defect, merging with a central scotoma on the left and a central scotoma for color on the right (fig. 8). The pupils were equally dilated and regular in outline and reacted well to light and in accommodation, but the reaction to light was not normally sustained. The extraocular movements were normal. The corneal reflexes were equally active on the two sides. There were no weaknesses of the face, palate or tongue.

Laboratory Data.—The urine was normal. The blood count revealed 78 per cent hemoglobin, 4,100,000 red cells, 8,200 white cells and a color index of 0.95.

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2. Traquair, H. M.: Bitemporal Hemiopia: The Later Stages and the Special Features of the Scotoma, Brit. J. Ophth. **1**:216, 281 and 377, 1917.

3. de Schweinitz, G. E.: Visual Phenomena in Pituitary Body Disease, Tr. Coll. Physicians, Philadelphia **37**:98, 1915.

4. Cushing, H., and Walker, C. B.: Distortions of the Visual Fields in Cases of Brain Tumor: Chiasmal Lesions, with Especial Reference to Bitemporal Hemianopsia, Brain **37**:341, 1915.

The serologic reactions of the blood were negative for evidence of syphilis. The spinal fluid was clear and colorless. The pressure was normal; there was less than 1 cell per cubic millimeter; the Wassermann reaction was negative, and the colloidal gold curve was normal.

Course.—Vision became progressively worse, and reexamination of the visual fields three weeks after the original study revealed extension of the field defects (fig. 9). An encephalogram was interpreted as inconclusive because of complete absence of filling of the ventricular system; the subarachnoid pathways were

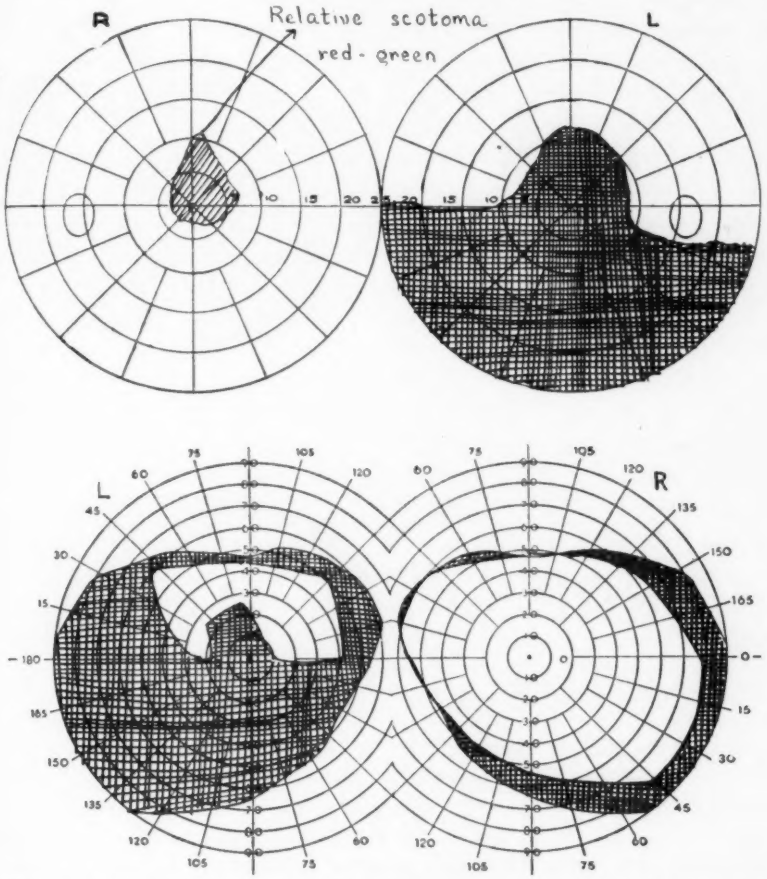

Fig. 8 (case 4).—Visual fields (15 mm. white test object for left eye; 5 mm. test object for right eye; distance, 330 mm.; daylight illumination), showing central scotoma merging with inferior altitudinal defect in the left eye and relative central scotoma in the right eye. Vision: right eye, 10/200 without glasses and 20/200 with glasses; left eye, perception of hand movements.

described as having "a fairly normal appearance." The patient was discharged on Sept. 17, 1942, with her condition unimproved. Subsequent information, obtained from another hospital, disclosed that a suprasellar meningioma had been found at operation.

Comment.—Here, too, the course was rapid over a period of three months, and headaches and visual loss were the outstanding symptoms. Optic nerve atrophy was not prominent, and the field defects were not of a hemianoptic type. Noteworthy in this case was the presence of an inferior altitudinopia in the left eye. Though tumor was suspected, a

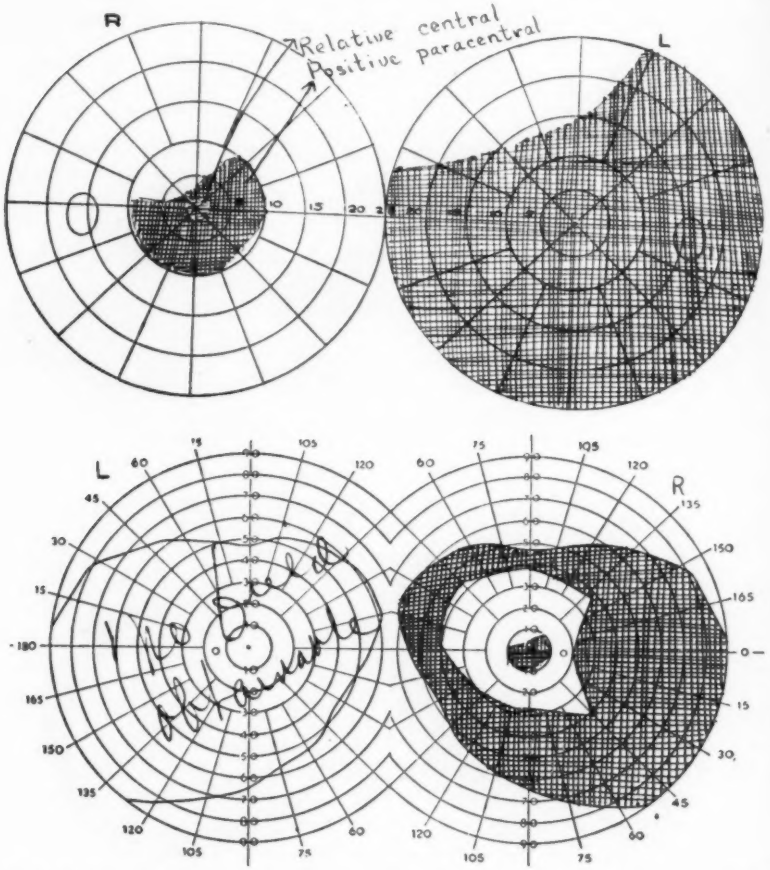

Fig. 9 (case 4).—Fields taken with 5 mm. test object at 330 mm., daylight illumination, three weeks after those in figure 8, showing expansion of defect to near obliteration of vision on the left (perception of hand movements) and definite central scotoma with peripheral constriction on the right. Vision in the right eye was 5/200 with and without glasses.

pneumoencephalogram gave inconclusive evidence, and the patient was discharged, only to have a suprasellar meningioma revealed at operation in another hospital. The rapid expansion of field defects is significant and indicates the need for speed in diagnosis and in neurosurgical intervention whenever central scotomas become manifest.

GENERAL COMMENT

The meningiomas in the suprasellar region have been described by Cushing [5] as arising from the meninges overlying the circumsellar venous sinuses. The need of more accurate localization of these meningiomas as to their site of origin from the tuberculum sellae was later appreciated by Cushing and Eisenhardt,[6] who also described in detail the common clinical features of these tumors. An early clinicopathologic report of a suprasellar meningioma was made by Stewart,[7] and it illustrates the clinical course of such a lesion prior to the advent of neurosurgical intervention. Dandy [8] and Cushing [5] both reported in 1922 their results in exploring the region of the optic chiasm. Subsequently, the operative accessibility of the suprasellar meningioma was emphasized by Holmes and Sargent [9] and by Cushing.[10] At the stage at which this tumor is more easily removable, it does not deform the sella turcica and does not cause symptoms of endocrine dysfunction. Consequently, the importance of correctly interpreting the fundus and, more especially, the alterations in the visual fields when these symptoms first appear in such cases is obvious.

As a basis for a clearer appreciation of the varied types of defects of the visual fields which may be caused by suprasellar meningiomas, a brief survey of the regional anatomy would not be amiss. Grossly, the optic nerve at the apex of the orbit passes through the optic canal, in which it lies above the ophthalmic artery. Intracranially, the optic nerves cross the internal carotid arteries dorsally before reaching the optic chiasm. At one time the optic chiasm was thought to have a rather constant position in relation to the base of the skull in the region of the chiasmatic sulcus. However, Schaeffer [11] has conclusively demonstrated that in the majority of instances the chiasm is in the neighbor-

5. Cushing, H.: The Meningiomas (Dural Endotheliomas): Their Source and Favored Seats of Origin, Brain **45**:282, 1922.

6. Cushing, H., and Eisenhardt, L.: Meningiomas Arising from the Tuberculum Sellae, Arch. Ophth. **1**:1 (Jan.); 168 (Feb.) 1929.

7. Stewart, J.: The Symptomatology of Tumors Involving the Hypophysis, Tr. A. Am. Physicians **14**:282, 1899.

8. Dandy, W. E.: Prechiasmal Intracranial Tumors of the Optic Nerves, Am. J. Ophth. **5**:169, 1922.

9. Holmes, G., and Sargent, P.: Suprasellar Endotheliomata, Brain **50**:518, 1927.

10. Cushing, H.: The Chiasmal Syndrome of Primary Optic Atrophy and Bitemporal Field Defects in Adults with a Normal Sella Turcica, Arch. Ophth. **3**:505 (May); 704 (June) 1930.

11. Schaeffer, J. P.: Some Points in the Regional Anatomy of the Optic Pathway, with Especial Reference to Tumors of the Hypophysis Cerebri and Resulting Ocular Changes, Anat. Rec. **28**:243, 1924.

hood of the diaphragma sellae, a site considerably posterior to the sulcus chiasmatis.

We are concerned in this paper chiefly with the defects of the visual fields which may result from the destructive action of a suprasellar meningioma on the primary visual pathways. Although it might be expected that such a lesion would produce constant and predictable alterations in the visual fields, owing to compression of the chiasm, it is not an uncommon experience to find that the expected changes in the visual fields, namely, symmetric bitemporal hemianoptic defects, are absent. However, the anatomic fact previously mentioned, that the optic chiasm is most often located in the neighborhood of the diaphragma sellae, may be correlated with the fact that the suprasellar meningiomas originate in the region of the tuberculum sellae, resulting in the inescapable conclusion that many, if not the majority of, such neoplasms will initially affect the prechiasmal portions of the visual pathway rather than the chiasm. This is sufficient to explain the tendency toward asymmetry of the temporal field defects and the absence of homonymous hemianoptic field defects, as well as the occurrence in some instances of altitudinal field defects. These observations are apparent in our cases and have been pointed out previously by Cushing and Eisenhardt.[6]

Our cases demonstrate in a striking manner the somewhat surprising and unexpected clinical observation that suprasellar meningiomas are in some instances associated with a syndrome which includes scotomatous field defects. These scotomas sooner or later join with a peripheral field defect to produce an expanding sector defect which ultimately results in blindness. Scotomatous field defects have been said to occur in about 15 per cent of cases of tumors of the pituitary gland,[12] have been described by Lillie[13] in cases of chiasmal arachnoiditis and are included as a feature of the Foster Kennedy syndrome.[14] However, a survey of the literature discloses that the relatively early appearance of central scotomatous field defects in cases of suprasellar meningioma has not been sufficiently emphasized. The central scotoma in such cases presents a difficult problem in diagnosis, often being attributed to retrobulbar neuritis or to multiple sclerosis rather than being considered as a possible sign of tumor in the suprasellar region. Of help in early differential diagnosis may be the outline of an incompletely developed scotoma in the central field. In addition to the out-

12. Alpers, B. J.: Diagnosis and Treatment of Pituitary Tumors, M. Clin. North America **26**:1679, 1942.

13. Lillie, W. I.: Prechiasmal Syndrome Produced by Chronic Local Arachnoiditis, Arch. Ophth. **24**:940 (Nov.) 1940.

14. Kennedy, F.: Retrobulbar Neuritis as an Exact Diagnostic Sign of Certain Tumors and Abscesses in the Frontal Lobes, Am. J. M. Sc. **141**:355, 1911.

line of the central field defect, which at times may be fully developed when the patient is first seen, an important characteristic of such fields is the peripheral indentation which is almost always present and which sooner or later merges with the central defect to produce an expanding sector defect in the visual field.

The relationship between the location of lesions involving the primary visual pathway and the type of central scotoma was reviewed by Walsh and Ford.[15] It was indicated by these authors that a unilateral, roughly circular, pericentral scotoma results from involvement of macular or papillomacular bundles. Hemianoptic scotomas, which are often associated with a contralateral peripheral field defect, were described by Traquair[16] as being a result of lesions at the junction of the optic nerve and the optic chiasm. Lesions of the posterior portion of the chiasm are said to cause bitemporal central and peripheral field defects. According to Walsh and Ford,[15] lesions at the posterior angle of the chiasm result in homonymous field defects, which centrally may include a contralateral temporal scotoma, either alone or combined with a homolateral pericentral scotoma.

Consideration previously given to the appearance of central scotomatous field defects as a manifestation of lesions in the region of the optic chiasm has been largely in connection with their occurrence as a result of tumors of the pituitary body. Attention was first directed to the existence of an expanding paracentral defect in cases of such tumors by Foerster.[17] In his comprehensive discussion of the ocular aspects of disorders of the pituitary body, de Schweinitz[18] reviewed the various hypotheses which have been offered to explain the pathogenesis of such central field defects. These explanations have variously emphasized the factors of pressure, traction,[19] individual variations in the composition of the chiasm[20] and the selective affinity of toxins for the macular fibers.[21] Traquair[2] expressed the belief that the

15. Walsh, F. B., and Ford, F. R.: Central Scotomas, Arch. Ophth. **24**:500 (Sept.) 1940.

16. Traquair, H. M.: An Introduction to Clinical Perimetry, St. Louis, C. V. Mosby Company, 1927.

17. Foerster: Beziehungen der Krankheiten des Nervensystems zum Sehorgan, in Graefe, A., and Saemisch, T.: Handbuch der gesamten Augenheilkunde, Leipzig, W. Engelmann, 1877, vol. 7, p. 116.

18. de Schweinitz, G. E.: Concerning Certain Ocular Aspects of Pituitary Body Disorders, Mainly Exclusive of the Usual Central and Peripheral Hemianopic Field Defects, Tr. Ophth. Soc. U. Kingdom **43**:12, 1923.

19. Fisher, J. H., in Discussion on Disease of the Pituitary Body, Proc. Roy. Soc. Med. **6**:53, 1913.

20. de Schweinitz, G. E., and Carpenter, J. T.: Ocular Symptoms of Lesions of the Chiasma, J. A. M. A. **44**:81 (Jan. 14) 1905.

21. Fuchs, E., in discussion on Benson: Case of Acromegaly, Brit. M. J. **2**: 949, 1895.

acuteness of pressure, with impairment of the vascular supply to the macular fibers, rather than the site of such pressure on the primary visual pathways is responsible for the occurrence of central scotomas. Generally accepted has been the significant and relevant physiologic fact that the macular fibers are relatively more susceptible to pressure and toxic factors than the rest of the visual fibers. This physiologic principle, together with the anatomic principle based on the studies of Schaeffer,[11] serves as a reasonable basis for understanding the early, and relatively frequent, occurrence of scotomatous field defects in patients having suprasellar meningiomas. Noteworthy in this connection is the conclusion reached by Henderson[22] and supported by Jefferson[23] that central scotoma due to adenoma of the pituitary body is indicative of a prefixed optic chiasm with retrochiasmal extension of the tumor. On the other hand, our observations in cases of suprasellar meningiomas, as well as the observations of Wagener and Love[24] in cases of tumors of Rathke's pouch, tend to favor the likelihood of a prechiasmal location in such cases.

In the prechiasmal syndrome, as observed in cases of suprasellar meningioma, the outstanding symptom is impairment of vision, which in many instances is rapidly progressive. Characteristically, the visual impairment affects the two eyes successively rather than simultaneously, and the time interval is usually measurable in weeks or months rather than in days. In the early stage, optic disks either appear to be within normal limits or show only slight pallor. Later, the optic disks will show more or less pronounced atrophy. The visual field defects are an early and conspicuous clinical feature in the syndrome produced by this tumor. As might be expected, the field defects tend toward the "bizarre" type, in which a combination of central and peripheral defects exists. There are wide variations as to the size, shape and position of these field defects. The occurrence of scotomatous field defects centrally is probably more frequent as an initial type of alteration than is commonly recognized, and explains the early rapid failure of vision. The accuracy of the surmise by Holmes and Sargent[9] in this connection is supported by the observations in our cases. Of similar significance is the case report by Hagedoorn,[25] in which a suprasellar meningioma was discovered to be responsible for a bitemporal central field defect. The asymmetry and incomplete

22. Henderson, W. R.: The Pituitary Adenomata, Brit. J. Surg. **26**:811, 1939.

23. Jefferson, G.: Extrasellar Extensions of Pituitary Adenomas, Proc. Roy. Soc. Med. **33**:433, 1940.

24. Wagener, H. P., and Love, J. G.: Fields of Vision in Cases of Tumor of Rathke's Pouch, Arch. Ophth. **29**:873 (June) 1943.

25. Hagedoorn, A.: The Chiasmal Syndrome and Retrobulbar Neuritis in Pregnancy, Am. J. Ophth. **20**:690, 1937.

character of such central scotomas, emphasized previously by Nettleship,[26] as well as their subsequent junction with peripheral temporal or altitudinal field defects, are sufficient in most cases to differentiate suprasellar tumors from retrobulbar neuritis. Peripheral bitemporal field defects unaccompanied with scotomas are the exception, rather than the rule, in cases of suprasellar meningiomas. Asymmetric, altitudinopic field defects are observed in some cases and are pathognomonic of a prechiasmal lesion. In contrast to this, it may be stated that homonymous hemianoptic field defects apparently never occur with suprasellar meningiomas.

We do not believe that it is possible on the basis of the visual field defects alone to differentiate between the various suprasellar lesions which may be responsible for such defects. In addition to meningiomas, other lesions in the suprasellar region that must be considered are adenomas of the pituitary gland, tumors of the hypophysial duct, gliomas of the optic chiasm, aneurysm and chronic chiasmal arachnoiditis. In their article dealing with visual field defects associated with tumors of the hypophysial duct, Wagener and Love [24] reported the occurrence of unilateral or bilateral central scotoma in 11 of 23 cases and complete amaurosis of one eye in 2 other cases.

Improvements are constantly being made in the differential diagnosis of various lesions which may produce a prechiasmal syndrome. However, vigilance on the part of the neurologist and ophthalmologist is essential in order to avoid the error of attributing scotomatous field defects associated with suprasellar tumors to multiple sclerosis or retrobulbar neuritis. The delays which result from such errors in diagnosis are particularly regrettable in cases of suprasellar meningioma because they so often lead to complete loss of vision or to growth of the tumor to such dimensions that its complete removal becomes extremely hazardous or impossible. Examination of the spinal fluid may be diagnostically valuable if it reveals a positive serologic reaction, an abnormal colloidal gold curve or an elevated total protein content. Cerebral arteriography may be of assistance in cases in which aneurysm is suspected. However, in our opinon, the pneumoencephalographic examination promises to be the most accurate means of establishing or excluding preoperatively the diagnostic possibility of a suprasellar tumor and should be done whenever a prechiasmal syndrome is evident.

SUMMARY AND CONCLUSIONS

The regional anatomy of the prechiasmal and chiasmal portions of the primary visual pathway is such as to make completely uniform

26. Nettleship, E.: Central Amblyopia as an Early Symptom in Tumor at the Chiasma, Tr. Ophth. Soc. U. Kingdom **17**:277, 1897.

alterations in the visual fields resulting from suprasellar lesions practically impossible. Four cases are described which demonstrate the early occurrence of scotomatous field defects in patients with suprasellar meningiomas. Characteristically, the scotomatous field defects in such cases become identifiable as a clue to the existence of a prechiasmal or chiasmal lesion by combining with a peripheral encroachment on the visual field at some point so as to produce an expanding sector defect.

In order to increase further efficiency in establishing an early diagnosis of suprasellar meningioma, we believe that the syndrome of atrophy of the optic nerve with bitemporal visual field defects, as previously emphasized, should be broadened to include the combination of optic nerve atrophy and expanding scotomatous field defects. In the absence of headaches and of deformities of the sella turcica, the combination of optic nerve atrophy with either bitemporal or scotomatous field defects is sufficient to warrant routinely the performance of complete examination of the spinal fluid and pneumoencephalography.

Since meningiomas are histologically benign tumors and since the suprasellar location of meningiomas is potentially accessible to the neurosurgeon, an early diagnosis of these tumors is distinctly to be desired.

255 South Seventeenth Street.

HYDROGEN ION CONCENTRATION OF THE AQUEOUS

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AND
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THE value of the glass electrode for determination of the hydrogen ion concentration in the vitreous [1] led to its use for similar investigations on the aqueous humor. The studies were indicated in view of the variations in the physiologic values cited in the literature. The differences were often due to the unavoidable errors inherent in the applied methods. Early reports on the p_H of the aqueous lacked reliability, mostly because of the escape of carbon dioxide during the procedure.[2] After Goldschmidt [3] and Baurmann [4] drew attention to this error, it was usually avoided by methods employed in the examination of similar biologic fluids of moderate buffering capacity.[5] In general the studies

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From the Department of Ophthalmology of Columbia University College of Physicians and Surgeons, and the Institute of Ophthalmology, Presbyterian Hospital.

1. von Sallmann, L.: Hydrogen Ion Concentration of the Vitreous in the Living Eye, Arch. Ophth. **33**:32 (Jan.) 1945.

2. (*a*) Foa, C.: La reazione dei liquidi dell'organismo col metodo elettrometrico, Arch. di fisiol. **3**:369, 1906. (*b*) Hertel, E.: Ueber die Bestimmung der Wasserstoffionenkonzentration des Kammerwassers, Arch. f. Ophth. **105**:421, 1921. (*c*) Nordenson, J. W.: Die Wasserstoffzahl des Kammerwassers bei Föten in verschiedenen Lebensaltern, Upsala läkaref. förhandl. **26**:55, 1921; abstracted, Zentralbl. f. d. ges. Ophth. **7**:515, 1922. (*d*) Scalinci, N.: Richerche sulla reazione dei liquidi oculari, Arch. di sc. biol. **6**:341, 1924. (*e*) Lehmann, G., and Meesmann, A.: Ueber das Bestehen eines Donnangleichgewichtes zwischen Blut und Kammerwasser bzw. Liquor cerebrospinalis, Arch. f. d. ges. Physiol. **205**:210, 1924. (*f*) Meesmann, A.: Ueber die Abhängigkeit des intraokularen Druckes von der Wasserstoffionenkonzentration, Arch. f. Augenh. **94**:115, 1924. (*g*) Schall, E.: Das Vorkommen der Nadi Oxydasen in den Gewebszellen des Auges in seinen verschiedenen Entwicklungstufen, Arch. f. Ophth. **115**:666, 1925. (*h*) Gala, A.: Ionenkonzentration des Kammerwassers und Glaskörpers bei normalen und glaukomatösen Augen, Časop. lék. česk. **64**:409, 1925; abstracted, Zentralbl. f. d. ges. Ophth. **15**:112, 1926.

3. Goldschmidt, M.: Beitrag zur Aetiologie der Cataracta diabetica, Ber. ü. d. deutsch. ophth. Gesellsch. **45**:191, 1925.

4. Baurmann, M.: Die Messung der H-Ionenkonzentration im Kammerwasser, Ber. ü. d. deutsch. ophth. Gesellsch. **45**:280, 1925; Ueber neue Messungen der H-Ionenkonzentration in menschlichen Kammerwasser, Arch. f. Ophth. **118**:36, 1927.

(Footnotes continued on next page)

were conducted along four lines—by the colorimetric method with indicators; by the calculation of the hydrogen ion concentration as a function of the carbon dioxide content; by the calculation of the hydrogen ion concentration on the basis of the accurately determined free carbon dioxide and sodium bicarbonate in the aqueous, and, finally, by direct potentiometric measurement with the hydrogen electrode.

The first method of estimating the hydrogen ion concentration, based on the color changes of indicators, is known to be insufficiently accurate, and the error due to the loss of carbon dioxide cannot be fully avoided. In addition, the small amount of test fluid available requires a dilution of the sample, and in a plasmoid aqueous the protein and salt error invalidates the results. Values of the p_H with a range of 6.9 (Schall [2g]) to 7.77 (Lehmann and Meesmann [2e]) were recorded with this technic.

The second method of measuring the p_H by its calculation as a function of an assumed carbon dioxide content of the aqueous was elaborated by Goldschmidt [3] and later by Kubik.[5e] They exposed the aspirated sample in a micro-hydrogen-electrode to a carbon dioxide tension of 40 to 50 mm. of mercury and estimated the p_H range by means of the Henderson-Hasselbalch formula.

The third method of estimation of the p_H, which was applied by Mawas and Vincent [5b] and by Kronfeld,[5d] consisted in its calculation from the accurately determined content of native carbon dioxide and sodium bicarbonate in the same sample of aqueous with the Henderson-Hasselbalch formula. Kronfeld obtained the figures for the calculations with precise methods of gas analysis. His results were in agreement with

5. (*a*) Salit, P. W.: A Study of the Acid-Base Equilibrium of the Ocular Humors, Biochem. J. **24**:596, 1930. (*b*) Mawas, J., and Vincent, M.: Note sur le p_H et la reserve alcaline de l'humeur aqueuse, abstracted, Ann. d'ocul. **162**:620, 1925. (*c*) Mawas, J.: Sur le p_H et la reserve alkaline de l'humeur aqueuse, abstracted, Skandinav. Arch. f. Physiol. **49**:184, 1926; Nouvelles recherches sur la concentration ionique et la reserve alcaline de l'humeur aqueuse à l'état normal et dans les états glaucomateux, Internat. Ophth. Cong. **1**:161, 1929. (*d*) Kronfeld, P.: Der Kohlensäuregehalt des Kammerwassers, Arch. f. Ophth. **118**:606, 1927. (*e*) Kubik, J.: Experimentelle und klinische Untersuchungen über die Wasserstoffionenkonzentration des Kammerwassers, Arch. f. Augenh. **98**:483, 1928. (*f*) Vincent, M.: Action de l'adrénaline en instillation dans le cul-de-sac conjunctival sur le propriétés physicochimiques de l'humeur aqueuse, Compt. rend. Soc. de biol. **98**:1284, 1928. (*g*) Tria, E., and de Simone, C.: Ricerche chimico-fisiche sui liquidi oculari (acqueo e vitreo), Arch. di sc. biol. **17**:274, 1932. (*h*) Bosa, F.: Il p_H nell'acqueo e nel cristallino dis animali paratireoprivi, Rassegna ital. d'ottal. **7**:613, 1938; abstracted, Zentralbl. f. d. ges. Ophth. **43**:162, 1939. (*i*) Taliercio, A.: La cataratta da raggi X, Ann. di ottal. e clin. ocul. **67**:104, 1939. (*j*) de Crecchio, A., and Brignola, D.: Riserva alcalina del sangue e p_H dell'acqueo nell'acidosi sperimentale, ibid. **67**:781, 1939. (*k*) Pignolosa, G.: Il p_H dell'umore acqueo nel coniglio nelle affezioni sperimentali di natura tuberculare dell'uvea, ibid. **69**:307, 1941; abstracted, Zentralbl. f. d. ges. Ophth. **48**:107, 1942.

those obtained by Baurmann,[4] who first conducted potentiometric measurements of withdrawn samples of the aqueous with the syringe hydrogen electrode. This fourth method was also selected by Mawas and Vincent,[6] Tria and de Simone,[5g] Bosa,[5h] Taliercio,[5i] Kurose,[6a] de Crecchio and Brignola [5j] and Pignolosa.[5k] Despite the application of the same method on the same experimental animal (rabbit), the p_H values ranged from 7.06 (Tria and de Simone) to 7.61 (Taliercio). It is evident that aqueous fluid, like other solutions with a relatively high content of carbon dioxide, is not well suited to determination of the p_H with the hydrogen electrode. The small quantity of the sample presents another difficulty. Moreover, the protein error of this method excludes its use for plasmoid aqueous.

The glass electrode was scarcely known when most of these reports were published, between 1921 and 1930. In subsequent years it was introduced for determination of the p_H of biologic fluids and tissues and was used extensively in various fields of medical research after the invention of the thermionic amplifier. Nevertheless, a preference for the hydrogen electrode was shown in the studies on the p_H of ocular fluids in the last decade. Except for the use of the glass electrode by Adams and Kerridge in 1930 [7] on the aspirated vitreous fluid and the investigation of this laboratory on the hydrogen ion concentration of the vitreous in vivo,[1] there have been no attempts to utilize the advantages of the glass electrode in ophthalmology. These advantages are fully described in the standard text by Dole [8] and are also reviewed briefly in the study on the p_H of the vitreous.[1]

THE GLASS ELECTRODE AND ITS USE

The electrode used in the present study was a modification of the capillary type originally described by MacInnes and Belcher [9] and improved by Michaelis.[10] It consisted of a straight microcapillary which was sealed into one arm of a U tube (figure). The tube was filled with freshly prepared quinhydrone–hydrochloric acid solution; a platinum wire, which was connected to a Beckman laboratory model potentiometer by means of a shielded lead, was immersed in the solution through the second arm of the U tube. The measuring capillary was made of Corning 015 electrode glass, with a slightly wider and heavier upper

6. Mawas and Vincent.[5b] Mawas.[5c] Vincent.[5f]

6a. Kurose, I.: Acta Soc. ophth. jap. **34**:217, 1930; cited by Crecchio and Brignola.[5j]

7. Adams, D. A., and Kerridge, P. T.: The Hydrogen Ion Concentration of the Vitreous, Brit. J. Ophth. **14**:397, 1930.

8. Dole, M.: The Glass Electrode, New York, John Wiley & Sons, Inc., 1941.

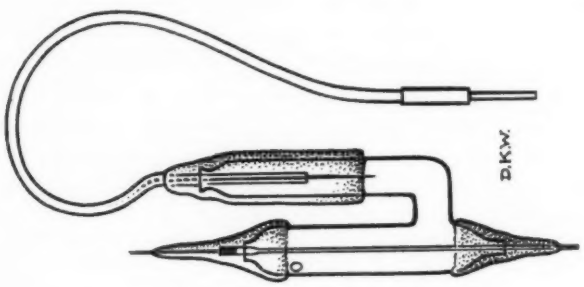
9. MacInnes, D. A., and Belcher, D.: A Durable Glass Electrode, Indust. & Engin. Chem. (Anal. Ed.) **5**:199, 1933.

10. Michaelis, L.: Glass Electrode with Galvanometer Reading, Science **83**: 212, 1936.

end, into which was cemented a short, stainless steel, 27 gage needle.[11] De Khotinsky cement was applied around the base of the needle to form a cone, which gave the needle firm support and permitted its insertion into a rubber sleeve used in the cleaning of the capillary and its standardization with buffers. The cement also covered the free extension of the capillary outside the U tube to prevent interference by a deviation film, as well as the other arm of the U tube, into which the platinum wire was inserted. Electrodes with a low zero correction, which gave a linear graph with buffers at a p_H of 4, 7 and 8, were selected. The electrodes were calibrated with a buffer solution prior to and after each determination. Readings on the aqueous were considered reliable only when the buffer solution read within 0.02 p_H unit after each experiment. The capillary was thoroughly rinsed with distilled water after each use and with a dilute solution of neutral soap after the determination on a plasmoid aqueous.

TECHNIC OF EXPERIMENTS

The anterior chamber was punctured with the needle, and aqueous was allowed to flow through the capillary until approximately half was drained. After the free end of the capillary was blocked with an agar–potassium chloride disk, the



Micro-glass-electrode used.

electrode was withdrawn; the opening of the needle was immediately closed with another agar–potassium chloride disk, and the electrode was then quickly transferred to a shielded constant temperature air chamber[12] in which the calomel half-cell electrode had been placed. Contact was made with the reference electrode by a fluid junction with a saturated solution of potassium chloride. The temperature of the latter was registered on an Anschütz thermometer, and that of the quinhydrone–hydrochloric acid solution in the U tube was measured with a thermocouple. With an appropriate warming of the electrode before use and with the regulation of the water bath in which the potassium chloride solution was kept, it was possible to obtain readings at a temperature of 36 to 37 C. in both solutions. The control readings of the buffer solution prior to and after determinations on the aqueous were carried out at approximately the same temperature.

11. The capillary electrodes were made by Frederick F. Anderson, of the department of pharmacology, Columbia University College of Physicians and Surgeons.

12. Dr. H. T. Clark, of the department of biochemistry, Columbia University College of Physicians and Surgeons, permitted the use of the constant temperature air chamber.

For the in vivo experiments general anesthesia was induced by the intraperitoneal injection of sodium pentobarbital, 40 mg. per kilogram of body weight. A suture was inserted at the border of the upper lid after injection of a small amount of 0.1 per cent nupercaine hydrochloride. The lid was retracted by means of the suture to expose the upper portion of the limbus. The rabbit was placed on an insulated operating board, which was fitted with glass supports for the electrode assembly. The needle of the properly warmed electrode was introduced into the anterior chamber and was held in place with a hard rubber clamp, which could be adjusted by a series of joints. After adequate drainage of the aqueous through the capillary, the distal end of the capillary was connected by an agar–potassium chloride bridge to the calomel half-cell. The operating board was placed in the constant temperature air chamber, and the electrodes were connected to the potentiometer by shielded leads. The electrodes were calibrated prior to and after each measurement of the aqueous under the conditions of the experiments. The same criteria for the reliability of the readings were applied as in in vitro experiments.

OUTLINE OF EXPERIMENTS

Chinchilla rabbits were selected as the experimental animals. In preliminary tests the temperature in the anterior chamber was measured by a suitably formed thermocouple at a room temperature of about 26 C. An average temperature of 36.6 C. was read when local anesthesia with 0.1 per cent nupercaine hydrochloride had been induced. The reading was slightly lower, 36.4 C., in rabbits under prolonged systemic anesthesia with sodium pentobarbital. These results of measurement of temperature in the anterior chamber compared well with those reported by Nelson[13] and Kokott.[14] Most of the p_H determinations were performed, therefore, at about 36.5 C. in a constant temperature air chamber. In a relatively large comparative series, the experiments were conducted at room temperature to obtain a conversion factor for the projected adaptation of the method for human subjects.

The study was first concerned with the influence of the various types of anesthesia on the p_H of the aqueous. For local anesthesia 1 drop of 0.1 per cent nupercaine hydrochloride (about p_H 5) was instilled three times at intervals of one minute in one eye; the same anesthetic was applied to the other eye over a period of one hour at intervals of fifteen minutes. In a small series of experiments anesthesia was induced with butacaine sulfate. For systemic anesthesia 40 mg. of sodium pentobarbital per kilogram of body weight was injected intraperitoneally. Measurement of the p_H of the aqueous of one eye took place as soon as the anesthesia was deep enough, that is, after eighteen to twenty-three minutes, and that of the other eye about one hour later. Additional experiments were conducted to study the effect of ether anesthesia and that of subcutaneous injections of morphine on the hydrogen ion concentration of the aqueous.

The diet of the rabbits consisted of oats and hay. Three rabbits received a diet rich in alkali for a period of five weeks, and the p_H values of the aqueous were collated with those of rabbits on the usual diet.

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13. Nelson, F.: Experimenteller Beitrag zur Frage der Temperatur in der vorderen Augenkammer, insbesondere bei Anwendung des Dampfkauters nach Wessely, Klin. Monatsbl. f. Augenh. (Beilagehft.) **78**:48, 1927.

14. Kokott, W.: Zur Frage der Kurzwellenbehandlung des Auges; Untersuchungen über die Erwärmung des Glaskörpers im Kaninchenauge bei Kurzwellenbestrahlung mit einem Röhrensender, Klin. Monatsbl. f. Augenh. **97**:448, 1936.

Another series of experiments pertained to the influence of a high protein content of the aqueous on its p_H. For this purpose vasodilators were introduced into the anterior chamber by iontophoresis, preliminary experiments having shown that this procedure greatly increased the proteins. Plasmoid aqueous was also produced by intravitreal infection with Staphylococcus aureus.

Finally, attempts were made to shift the hydrogen ion concentration by introducing anions from a solution of sodium bicarbonate by ion transfer. In relation to previous studies on iontophoresis with antibiotic substances, the p_H of the aqueous was also determined after the iontophoretic application of sulfonamide compounds and penicillin.

RESULTS AND COMMENT

The p_H of the aqueous changed significantly with the selected type of anesthesia. It varied with nupercaine anesthesia from 7.48 to 7.59, with an average of 7.53, for 24 determinations on the normal eyes of

TABLE 1.—*p_H of the Aqueous of Rabbits with the Eye Under Local Anesthesia*

With Nupercaine Hydrochloride At Body Temperature			At Room Temperature			With Butacaine Sulfate At Room Temperature		
	Brief Anesthesia			Brief	Prolonged			
Rabbit Number	OD	OS	Rabbit Number	Anesthesia OD	Anesthesia OS	Rabbit Number		Control Eyes (Nupercaine)
1	7.58	7.58	15	7.67	7.68	25 OD	7.54	OS 7.54
2	7.49	7.52	16	7.66	7.76	26 OD	7.54	OS 7.55
3	7.55	7.57	17	7,65	7.70	27 OD	7.58	
4	7.49	7.49	18	7.58	7.70	OS	7.59	
5	7.52	7.54	19	7.64	7.69			
6	7.53	7.53	20	7.52	7.60	Average	7.56	7.545
7	7.50	7.55	21	7.63	7.66			
8	7.49	7.50	22	7.64	7.69			
9	7.50	7.54	23	7.65	7.70			
10	7.59	7.55	24	7.66				
11	7.59	....						
12	7.50	....	Average	7.63	7.677			
13	7.53	....						
14	7.48	....						
Average	7.53							

rabbits at a temperature of 36.5 C. and from 7.52 to 7.67, with an average of 7.63 for 10 eyes at a temperature of 23 C. (table 1). The values were obtained after the local anesthetic had been instilled three times, as described in the technic. The aqueous became on an average 0.047 p_H unit more alkaline (9 experiments) after continuation of the topical anesthesia for about one hour. When both eyes of the same animal were treated identically, the average difference between the two eyes was 0.02 p_H unit. Two local anesthetics were used, 0.1 per cent nupercaine hydrochloride, which strongly inhibits mitosis and cell migration, according to Smelser and Ozanics,[15] and 2 per cent butacaine sulfate, with its moderate effect on cell division.[16] No differences between

15. Smelser, G. K., and Ozanics, V.: Effect of Local Anesthetics on Cell Division and Migration Following Thermal Burns of Cornea, Arch. Ophth. **34:** 271 (Oct.) 1945.

these two anesthetics were noted, however, in respect to their influence on the p_H readings of the aqueous.

The values at 36.5 C. were in close agreement with those obtained electrometrically on the aqueous of rabbits by Baurmann [4] (p_H 7.5 to 7.52) and the calculated figures of Kronfeld and others. The authors, with the exception of Kubik [5e] and Sano,[16] did not specify the type of anesthesia. Whereas Kubik did not observe any effect of local cocaine anesthesia on the p_H of the aqueous, Sano reported changes in the p_H after instillation of various anesthetics and ophthalmotherapeutic solutions. In contrast to the present results, he found that cocaine decreased and that tutocaine and nupercaine increased the hydrogen ion concentration. It is doubtful whether the colorimetric method which he applied is appropriate for such investigations.

In view of the studies of Friedenwald and Buschke [17] on the depressing effect of systemically administered epinephrine on the migration of the corneal epithelium and the similar experiences of Smelser [18] with its topical application, the p_H of the aqueous was determined after the repeated instillation of epinephrine hydrochloride in 1: 1,000 dilution. Previous studies, with conflicting results were made by Sano [16] with an indicator method and by Vincent [5f] with the hydrogen electrode. The former reported an increase and the latter a significant decrease in the hydrogen ion concentration after instillation. In the present study the technic of Vincent was followed; that is, the solution was instilled three times at ten minute intervals. Table 2 indicates that the values obtained were within the p_H range of the control eyes and were at variance, therefore, with aforementioned reports.

Despite the various effects of anesthetics and epinephrine on mitosis and migration of epithelium cells described by Buschke and his associates,[19] Smelser and Ozanics [15] and Friedenwald and Buschke,[17] the p_H of the aqueous shifted in the same direction and degree with nupercaine hydrochloride and butacaine sulfate and remained unaffected with epinephrine. Nevertheless, the more alkaline readings of the aqueous

16. Sano, T.: Experimentelle Studien über den Einfluss der pupillomotorischen Mittel auf die Wasserstoffionenkonzentration des Augenkammerwassers, Acta Soc. ophth. jap. **36**:1105, 1932; abstracted, Zentralbl. f. d. ges. Ophth. **28**:473, 1933; Experimentelle Studien über den Einfluss der verschiedenen Augentrofmittel auf die Wasserstoffionenkonzentration des Augenkammerwassers, Acta Soc. ophth. jap. **36**:1837, 1932; abstracted, Zentralbl. f. d. ges. Ophth. **29**:86, 1933.

17. Friedenwald, J. S., and Buschke, W.: The Influence of Some Experimental Variables on the Epithelial Movements in Healing of Corneal Wounds, J. Cell. & Comp. Physiol. **23**:95, 1944.

18. Personal communication to the authors.

19. Buschke, W.; Friedenwald, J. S., and Fleischmann, W.: Studies on the Mitotic Activity of the Corneal Epithelium, Bull. Johns Hopkins Hosp. **73**:143, 1943.

of rabbits with local anesthesia, especially after prolonged topical anesthesia, can best be explained by the increase in permeability of the cornea due to epithelial damage. In their studies on the gaseous exchange through the cornea, Fischer [20] and Redslob and Tremblay [21] found that the cornea had a unidirectional permeability for carbon dioxide and oxygen. Fischer assumed that carbon dioxide passed through the cornea from the aqueous into the surrounding atmosphere and expressed the belief that the epithelium is a partial barrier for these two gases, since they pass through the normal epithelium to a moderate degree. The escape of carbon dioxide facilitated by the enhanced permeability of the cornea with topical anesthesia, therefore, may be the cause of the relative alkalinity.

TABLE 2.—*p_H of the Aqueous* After Instillation of Epinephrine, Injection of Morphine and Use of Alkali-Rich Diet*

Procedure	Rabbit No.	Treated Eye	Control Eye
Epinephrine hydrochloride (1:1,000), 2 drops instilled every ten minutes for ½ hour	28	7.63	7.62
	29	7.65	7.67
	30	7.60	7.65
Average		7.63	7.65
Morphine sulfate, 10 Mg./Kg. subcutaneously	31	7.66	7.66
	32	7.42†	7.51
	33	7.60	7.62
	34	7.64	7.64
Average		7.58	7.607
		OD	OS
Alkali-rich diet	35	7.64	‡
	36	7.65	‡
	37	7.59	7.64
Average		7.63	

* Determined with local nupercaine anesthesia at room temperature.
† Deep depression of respiration.
‡ Readings not reliable.

General anesthesia induced with a barbiturate had the converse effect on the hydrogen ion concentration of the aqueous humor (table 3). The average of 4 readings recorded after a brief general anesthesia with sodium pentobarbital was 7.495, but the p_H shifted to the acid side, 0.04, with the continuation of the anesthesia. When the in vivo technic was used, the average p_H was 7.44 about twenty minutes after the injection of sodium pentobarbital (7 eyes) and 7.4 about one hour after the injection (8 eyes). Except for 1 eye, the readings at a

20. Fischer, F. P.: Ueber den Gasaustausch der Hornhaut mit der Luft, Arch. Augenh. **120**:146, 1930.

21. Redslob, E., and Tremblay, J. L.: Etude sur les échanges gaseux a la surface de l'oeil, Ann. d'ocul. **170**:415, 1933.

later period indicated a change to the acid side as the depth and the duration of anesthesia increased. This shift was probably due to the usually pronounced depression of the respiratory center and the ensuing accumulation of carbon dioxide.

In vivo experiments gave slightly less alkaline readings than experiments on aspirated aqueous with the animal under pentobarbital anesthesia. This difference can be accounted for by the fact that a deeper anesthesia, and therefore a greater depression of the respiratory center, was necessary in the in vivo experiments to restrain the animal from undue movements. Another possible explanation is that of the escape of carbon dioxide from the measuring capillary in

TABLE 3.—*p_H of the Aqueous of Rabbits Under General Anesthesia*

With Sodium Pentobarbital												With Ether
In Vivo			In Vitro						In Vitro			
			At Body Temperature			At Room Temperature			At Room Temperature			
Rabbit No.	p_H OD Determined 18-23 Min. After Initiation of Anesthesia	p_H OS Determined 45-120 Min. After Initiation of Anesthesia	Rabbit No.	p_H OD Determined 10-35 Min. After Initiation of Anesthesia	p_H OS Determined 30-60 Min. After Initiation of Anesthesia	Rabbit No.	p_H OD Determined 10-20 Min. After Initiation of Anesthesia	p_H OS Determined 17-60 Min. After Initiation-of Anesthesia	Rabbit No.	p_H OD Determined 5 Min. After Initiation of Anesthesia	p_H OS Determined 15 Min. After Initiation of Anesthesia
38	7.44	7.39	47	7.48	7.46	51	7.59	7.53	56	7.53	7.54
39	7.47	7.41	48	7.49	7.43	52	7.60	7.54	57	7.59	7.57
40	7.43	7.39	49	7.48	7.45	53	7.56	7.46*	58	7.58	7.48*
41	7.43	7.38	50	7.53	7.48	54	7.63	7.49	59	7.55	
42	7.46	7.43				55	....	7.50			
43	7.44	7.44	Aver.	7.495	7.455				Aver.	7.56	7.53
44	7.42	....				Aver.	7.595	7.50			
45	....	7.41									
46	....	7.38									
Aver.	7.44	7.40									

* Asphyxia.

the brief period between the withdrawal of the needle and its blocking with an agar–potassium chloride disk in the in vitro technic. This explanation was disproved by the fact that the omission of the disk did not result in an increase in the p_H.

Whereas many investigators have found that systemic anesthesia induced with the barbiturates has little influence on the tissue and blood, the present study shows that this observation cannot be extended to a body fluid like the aqueous with a moderate buffering capacity. Experiments with ether anesthesia (7 eyes) supported the theory that the accumulation of carbon dioxide was operative in a shift of the p_H to the acid side, since a similar increase in the hydrogen ions was observed only when the animal had sustained a period of asphyxia (rabbit 58). An analogous trend was noted in a small series of

experiments with the subcutaneous injection of 10 mg. of morphine sulfate per kilogram of body weight, as in 1 of the 4 rabbits in which the respiration was obviously impaired the injection resulted in an increase in the hydrogen ion concentration of the aqueous (table 2).

In conclusion, the physiologic values of the p_H of the aqueous in rabbits can be considered in the range of 7.44 to 7.49, that is, at the beginning phase of general anesthesia with a barbiturate and before marked depression of the respiratory center has occurred. Both local and systemic anesthesia alter the p_H of the aqueous, especially when prolonged. It is desirable, therefore, to specify in reports on the p_H of the aqueous the type of anesthesia employed. Conclusions based on the effect of barbiturate anesthesia on the p_H of the aqueous can be extended to the vitreous; it is highly probable that the physiologic p_H of the vitreous, which could be measured only in vivo with deep barbiturate anesthesia, is more alkaline (p_H 7.1 to 7.2) than has previously been reported (p_H 7.0 to 7.1 [1]). A more detailed discussion of the relation of the hydrogen ion concentration of the aqueous and vitreous humors will be included in a later study on the p_H of both fluids of the same eye.

Kronfeld [5d] in a study on the carbon dioxide content of the aqueous in rabbits reported that it was influenced by the amount of alkali in the diet. He found that food rich in alkali led to an increase of the carbon dioxide content of the aqueous from 56.1 to 61.5 volumes per cent. An alkali-rich diet, similar to that used by Kronfeld, and consisting of leafy vegetables and bread, did not cause any noticeable change in the p_H of the aqueous (table 2). The values obtained in 4 determinations (3 rabbits) did not differ significantly from those obtained on animals with the usual oats and hay diet.

For the study of the relation of the protein content of the aqueous to its p_H, a considerable increase of protein was achieved by the iontophoretic introduction of histamine and physostigmine. One hour after an aqueous solution of 0.1 per cent histamine phosphate and 0.1 per cent physostigmine salicylate was applied for one minute at 1 milliampere from the anode, a sample of aqueous was withdrawn and the protein content determined with a modified nephelometric adapter in a Coleman spectrophotometer. It varied from 250 to 341 mg. per hundred cubic centimeters, with an average of 277 mg. per hundred cubic centimeters for 11 samples. Determinations on this plasmoid aqueous revealed that its p_H did not differ from that of untreated (control) eyes with a protein content of 20 to 50 mg. per hundred cubic centimeters (table 4).

It was previously reported that an experimental intravitreal infection, produced by the injection of a mannitol-fermenting strain of Staph. aureus and of Diplococcus pneumoniae, type III. was accompanied

in an early stage with a local acidosis of the vitreous, with p_H readings between 5.7 and 6.35. A similar staphylococcic infection of the vitreous caused, however, only a slight increase (0.07 p_H unit) in the hydrogen ion concentration of the aqueous. Its extent corresponded roughly to the development of the clinical signs of inflammation in the anterior segment. In table 4 are reported the measurements which were made twenty-four hours, forty-eight hours and one week after the infection. As determinations of the p_H were not made on the vitreous of these eyes, but are planned for a later study, the relation between the respective values of the hydrogen ion concentration in the two intraocular fluids will not be dealt with here. It is apparent, however, that the p_H of the aqueous is but little affected by the coexistence with an acute infection of the vitreous.

Meesmann [2f] did not observe a change in the p_H of the aqueous associated with inflammation, whereas Pignolosa [5k] reported an increase

TABLE 4.—*p_H of the Aqueous* With an Increase in Protein*

Due to Vasodilators Both Eyes Treated			Due to Vitreal Infection with Staphylococcus Aureus								
Rabbit No.	OD	OS	Rabbit No.	24 Hr. After Infection	Control Eye	Rabbit No.	48 Hr. After Infection	Control Eye	Rabbit No.	One Week After Infection	Control Eye
60	7.51	7.51	63	7.47	7.58	66	7.46	7.50	69	7.41†	7.57
61	7.54	7.52	64	7.42	7.48‡	67	7.52	7.53	70	7.57	7.58
62	7.53	....	65	7.47	7.59	68	7.45	7.58	71	7.38†	7.43‡
Aver.	7.53	7.515	..	7.45	7.55	..	7.48	7.54	..	7.45	7.53

* Determined with local nupercaine anesthesia at room temperature.
† Pronounced hyperemia and swelling of iris.
‡ Scarred and vascularized cornea.

of the hydrogen ion concentration of the aqueous of rabbits with an experimental tuberculous infection of the uvea and expressed the belief that the development of a complicated cataract, which he observed in many instances, was possibly dependent on this shift. Although this author's observations are in accord with those reported here, his results are not conclusive because of the use of the hydrogen electrode with a plasmoid aqueous.

Mawas [5c] suggested that local, noncompensated acidosis associated with certain types of glaucoma might be counteracted by ionization with alkalis. To our knowledge, this suggestion has not led to investigations on the influence of iontophoresis on the p_H of intraocular fluids. In the present study, such measurements were carried out after the iontophoretic introduction from the cathode of a 5 per cent solution sodium bicarbonate applied at a strength of 2 milliamperes for ten minutes and a 5 per cent solution of soduim sulfadiazine and 5,000 Oxford units of sodium penicillin per cubic centimeter applied

at a strength of 2 milliamperes for five minutes. Under the conditions of the experiment, none of the introduced anions caused a significant change in the p_H of the aqueous (table 5). It is probable

TABLE 5.—*p_H of the Aqueous* After Iontophoresis*

Procedure	Rabbit No.	Treated Eye	Control Eye
5 per cent sodium bicarbonate, 2 ma., 10 minutes.....	72	7.68	7.66
	73	7.6-7.3	7.62
		Drifting	
	74	7.61	7.60
	75	7.64	7.69
Average.....		7.64	7.64
5 per cent sodium sulfadiazine, 2 ma., 5 minutes.....	76	7.62	7.62
	77	7.57	7.57
	78	7.57	7.58
Average.....		7.59	7.59
Sodium penicillin, 5,000 Oxford units per cc., 2 ma., 5 minutes	79	7.64	7.60
	80	7.72	7.68
	81	7.62	7.64
	82	7.61	7.61
	83	7.62	7.63
Average.....		7.64	7.63

* Determined with local nupercaine anesthesia at room temperature.

that the ionic equilibrium was retained by a compensatory egress of anions by various channels under the influence of the impressed voltage.

SUMMARY

1. A capillary glass electrode was designed to determine the hydrogen ion concentration of the aqueous of rabbits in vitro and in vivo at a physiologic temperature.

2. With this electrode the average physiologic p_H of the rabbit aqueous was 7.53 with brief local anesthesia and between 7.44 and 7.49 in the initial stage of systemic anesthesia.

3. Prolongation of topical anesthesia was followed by a slight increase in the p_H. Lengthy general anesthesia led to a significant decrease in the p_H. A similar shift to the acid side was noted after asphyxia during anesthesia with ether and after pronounced depression of the respiratory center with morphine.

4. Ectogenous infections of the vitreous with Staph. aureus caused a moderate shift of the hydrogen ion concentration of the aqueous to the acid side.

5. A diet rich in alkali, an increase in the protein of the aqueous and the iontophoretic introduction of anions had no noticeable effect on the p_H of the aqueous.

630 West One Hundred and Sixty-Eighth Street.

TREATMENT OF CHRONIC BLEPHAROCONJUNCTIVITIS WITH PENICILLIN OINTMENT

Report of Twenty-Five Cases

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THE HISTORY of penicillin is essentially the story of two developments. The first was Fleming's accidental discovery of the inhibitive effect of Penicillium notatum on staphylocci in vitro.[1] Clutterbuck, Lowell and Rainstrick, stimulated by Fleming's study, attempted to extract penicillin in 1932, but their efforts were largely unsuccessful.

Second, through the extraction method, involving the passing of impure acid penicillin broth from the watery solution into an organic solvent (ether or amyl acetate) and the subsequent passing of the purified agent again into water (shaken with alkali), Florey and his colleagues showed how to obtain sufficient penicillin in a relatively pure form and demonstrated its clinical use.[2]

METHOD OF ASSAY

As the exact constitution of penicillin has not yet been worked out, the material cannot be standardized by chemical means. However, the success of research largely depended on a reliable procedure for assay. The method adopted was worked out by Dr. Norman Heatly, whereby penicillin is assayed by its biologic effect. The Florey (Oxford) unit—the amount of penicillin as compared with an arbitrary standard which completely inhibits the growth of a test strain of Staphylococcus aureus—is still used and probably will hold its place until a chemically standardized product is available. In Florey's original material there were 40 to 50 units per milligram (Abraham and others [3]). Material which runs to 1,000 units per milligram has now been prepared.

TOXICITY AND ANTIBACTERIAL ACTION

The relative lack of toxicity of penicillin for most tissues was apparent to Fleming [1] and to Florey and his associates.[2] Further studies carried

1. Fleming, A.: On the Antibacterial Action of Cultures of Penicillium, with Special Reference to Their Use in Isolation of B. Influenzae, Brit. J. Exper. Path. **10**:226 (June) 1929.

2. Chain, E.; Florey, H. W.; Gardner, A. D.; Heatley, N. G.; Jennings, M. A.; Orr-Ewing, J., and Saunders, A. G.: Penicillin as a Chemotherapeutic Agent, Lancet **2**:226 (Aug. 24) 1940.

3. Abraham, E. P., and others: Further Observations on Penicillin, Lancet **2**:177 (Aug. 16) 1941.

out by Herrell and his associates [4] revealed that penicillin was a highly antibacterial substance and had low toxicity as measured by tissue culture methods.

In evaluating the action of penicillin against infection in vivo, the anatomic site that lends itself best to treatment must be determined. Therefore, in treatment of ocular infections, the first step is to determine the concentration reached in the cornea by the various means of administration. Although von Sallmann and associates [5] have shown that penicillin enters the aqueous humor of the normal eye after application of the substance in a cup bath to the eye for five minutes, the present biologic methods of estimating the concentration of the drug make such a determination in corneal tissue most difficult. Bellows [6] has published his results on determination of the concentration of penicillin in the cornea and the conjunctiva. Later, von Sallmann and his associates [7] found that a 0.25 per cent solution of penicillin sodium penetrated the anterior segment of the eye. The local application of penicillin proved to be more effective than intramuscular administration in the treatment of corneal infections due to a gram-negative bacillus (Leopold, Holmes and LaMotte [8]).

BACTERIAL SENSITIVITY

The degree of sensitivity of the species of the infecting organism to the drug is but one of a number of factors which influence the therapeutic result. Also of importance are initial differences in sensitivity of strains of the same species to the selected compound and subsequent differences developing during the period of treatment, due in part to the maturation of the bacteria. Other factors are the number and the virulence of the infecting organisms, the amount of protein breakdown products or pus and the quality of the cellular and humoral body defenses.

4. Heilman, D. H., and Herrell, W. E.: Comparative Antibacterial Activity of Penicillin and Gramicidin: Tissue Culture Studies, Proc. Staff Meet., Mayo Clin. **17**:321 (May 27) 1942; Comparative Bacteriostatic Activity of Penicillin and Gramicidin, abstracted, J. Bact. **43**:12 (Jan.) 1942. Herrell, W. E.; Nichols, D. R., and Heilman, D. H.: Penicillin: Its Usefulness, Limitations, Diffusion and Detection, with Analysis of 150 Cases in Which It Was Employed, J. A. M. A. **125**:1003 (Aug. 12) 1944.

5. von Sallmann, L.; Meyer, K., and Di Grandi, J.: Penetration of Penicillin into the Eyes, Arch. Ophth. **31**:1 (Jan.) 1944.

6. Bellows, J. G.: Penicillin Therapy in Ocular Infections, Am. J. Ophth. **27**:1206 (Nov.) 1944.

7. von Sallmann, L.; Meyer, K., and Di Grandi, J.: Experimental Study on Penicillin Treatment of Ectogenous Infection of Vitreous, Arch. Ophth. **32**:179 (Sept.) 1944.

8. Leopold, I. H.; Holmes, L. F., and LaMotte, W. O., Jr.: Local Versus Systemic Penicillin Therapy of Rabbit Corneal Ulcer Produced by Gram-Negative Rod, Arch. Ophth. **32**:193 (Sept.) 1944.

Some of these factors cannot be evaluated at all, and others require time-consuming tests. The complication of crossed mixed infection in ocular diseases is not so important as in general infections but may retard recovery. There is evidence suggesting that with the elimination of a virulent penicillin-sensitive organism in a given case the remaining, nonsensitive organisms are not very pathogenic.

More than twenty bacteria clinically sensitive to penicillin have been described. The following penicillin-sensitive pathogens are encountered in ocular diseases: the alpha, beta and gamma streptococci; Staphylococcus aureus and Staphylococcus albus; Neisseria gonorrheae; Neisseria intracellularis; Corynebacterium diphtheriae (mitis); Clostridium welchii; Actinomyces bovis; Treponema pallidum, and diphtheroids. Grouped according to sensitivity to penicillin, the most highly sensitive organisms are the (beta) hemolytic streptococci, the gonococcus and some strains of staphylococci. The viridans (alpha) type of streptococcus, the nonhemolytic (gamma) streptococcus and the remaining organisms are less sensitive. Staphylococci vary from extreme sensitivity to extreme resistance.

Although gram-negative rods are insensitive to the action of penicillin, a few species are moderately susceptible, and still others exhibit a slight degree of sensitivity.[8] Abraham and his co-workers [8] showed that Salmonella enteritidis (gartneri) was inhibited by a 1:20,000 dilution, and Salmonella typhi by a 1:10,000 dilution of a solution of penicillin which inhibited a culture of Staph. aureus when diluted 1:1,000,000.

In the experience of Fleming [1]; Hobby, Meyer and Chaffee [9]; McKee and Rake,[10] and von Sallmann and Di Grandi,[11] members of the Friedländer group of bacilli have been insensitive to penicillin. However, Smith [12] showed that variation in strains is remarkable in this group, and the sensitive strains which he encountered included some of type A. A similar situation was reported by Churchman [13] in that among gram-negative rods, on the whole insensitive to the action of methylrosaniline chloride, an occasional strain may be encountered.

Penicillin should be given preference also in the treatment of infections due to streptococci and sensitive staphylococci. Some strains of

9. Hobby, G. L.; Meyer, K., and Chaffee, E.: Activity of Penicillin in Vitro, Proc. Soc. Exper. Biol. & Med. **50**:277 (June) 1942.

10. McKee, C. M., and Rake, G.: Biological Experiments with Penicillin, J. Bact. **43**:645 (May) 1942.

11. von Sallmann, L., and Di Grandi, J.: Simultaneeous Local Application of Penicillin and Sulfacetimide, Arch. Ophth. **32**:190 (Sept.) 1944.

12. Smith, L. D.: The Bacteriostatic Agent of Penicillium Chrysogenum, J. Franklin Inst. **254**:396, 1942.

13. Churchman, J. W.: Further Studies on Behaviour of Bacteria Toward Gentian Violet, J. Exper. Med. **33**:569 (May) 1921.

staphylococci are resistant to penicillin. If the resistant bacteria were responsible for the virulence of the infection, penicillin therapy was less beneficial.

One of the requirements for successful treatment of bacterial infections with penicillin is limitation of its use to infections due to pathogens which are known to be susceptible. Therefore great care has to be exercised in appraising the value of penicillin in a given ocular infection. The unavoidable error of clinical observation is high. Many ocular diseases are self limiting. Any therapeutic agent employed in such a disease may be credited erroneously with expediting recovery or producing a cure. Conversely, the possibility of desensitivity of a strain of staphylococci found in the eyelids and conjunctiva by previous penicillin therapy has to be considered.

PREPARATION AND DOSAGE

Through the large scale American production, penicillin is supplied in sealed glass bottles as a sodium salt. It has a brown or yellow granular appearance and is extremely soluble in water and in saline or dextrose solution. However, the material is unstable in air and very hygroscopic, its potency being impaired by heat and in acid mediums.[13a] Therefore the sealed ampules must be preserved in the refrigerator until used. A day's dose made up in the proper solution may be safely kept in the cold container.

In topical application the Committee on Chemotherapy has recommended solutions containing 250 or 500 units per cubic centimeter. However, no untoward effect has been observed from the use of concentrations far greater than those mentioned, and very possibly better results will be obtained with the use of stronger solutions. Significantly, it is not the purpose of this paper to discuss the unsettled question of adequate penicillin dosage. Excellent results have been reported with relatively small quantities of the material but this does not rule out the need of larger doses in some cases (Herrell,[14] Bloomfield and associates[15]).

Owing to the impractibility of continuous therapy with solutions of penicillin in overseas units, with the instability at room temperatures, another form of preparation of the substance was indicated in order to

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13a. However, the material is unstable in air and very hygroscopic, its potency being impaired by heat and in acid mediums (Abraham, E. P.; Chain, E., and Holiday, E. R.: Purification and Some Physical and Clinical Properties of Penicillin, Brit. J. Exper. Path. **23**:103 [June] 1942).

14. Herrell, W. E.: The Clinical Use of Penicillin: An Antibacterial Agent of Biologic Origin, J. A. M. A. **124**:622 (March 4) 1944.

15. Bloomfield, A. L.; Kirby, W. M. M., and Armstrong, C. D.: A Study of "Penicillin Failures," J. A. M. A. **126**:685 (Nov. 11) 1944.

obtain prolonged therapeutic action of penicillin of sufficient concentration in the eyes and at the same time to preserve its potency for a moderately long period at room temperature. After various unsuccessful research studies were made with the idea of incorporating some preservative, it was decided to dissolve the penicillin in an ophthalmic water-soluble ointment. Thus, a soluble ointment as a base for the penicillin was prepared, consisting of wool fat U.S.P., 17 per cent; white petrolatum U.S.P. 51 per cent; distilled water, 5 per cent, and light liquid petrolatum U.S.P. 22 per cent. In the additional 5 per cent of distilled water 100,000 Oxford units of penicillin was dissolved and then incorporated into 7 Gm. of the ointment. A small bead of penicillin ointment tested in vitro on a nutrient agar cup plate seeded with staphylococci produced an inhibition ring 50 mm. in diameter. After five weeks at room temperatures in the tropics, the penicillin ointment produced an inhibition ring 44 mm. in diameter. With this result, it is reasonably safe to state that penicillin ointment for all practical purposes is stable for at least one month at room temperatures. These results are in accord with those of Keyes,[16] who has gone further and obtained a penicillin ointment which has a stability of six months, the ointment however, being kept in a commercial refrigerator.

The stronger penicillin ointment was decided on in order to determine the efficiency of a more potent single daily application of penicillin. Such a method of administration had an advantage, as the patients were combat troops. After repeated quantitative measurements, it was determined that each application contained 700 to 800 Oxford units.

Florey and Florey,[17] Cashell,[18] Bellows [6] and others reported encouraging results in treating infections of the border of the lid and the conjunctiva with ointments containing penicillin.

Although all types of ocular infection were treated, this paper will limit itself to a report on a series of 25 cases of chronic blepharoconjunctivitis.

REPORT OF CASES

CASE 1.—This case is typical of 7 cases.

A man aged 35 reported on Dec. 10, 1944, complaining of scaling of both eyelids and bilateral irritative conjunctivitis. Intense lacrimation had interfered with his duties in the Air Corps for three months. At another base, prior to transfer to this hospital, he had received a course of treatment with 5 per cent ophthalmic sulfathiazole ointment, with only temporary relief. Vision was 20/20 in each eye, with ability to read Jaeger type 1. Examination revealed severe chronic blepharoconjunctivitis, with scales on the upper and lower lids

16. Keyes, J. E. L.: Penicillin in Ophthalmology, J. A. M. A. **126**:610 (Nov.) 1944.

17. Florey, M. E., and Florey, H. W.: General and Local Administration of Penicillin, Lancet **1**:387 (March 27) 1943.

18. Cashell, G. T. W.: Treatment of Ocular Infections with Penicillin, Brit. M. J. **1**:420 (March 25) 1944.

of both eyes and an injected palpebral and bulbar conjunctiva, with annoying lacrimation. Culture revealed Staph. aureus. Single daily applications of penicillin ointment was instituted. Within forty-eight hours absence of scales was noted, and the conjunctival irritation was less intense. Twenty-four hours later the redness and swelling of the lid margins had subsided to a notable degree. After one week's treatment all signs and symptoms had disappeared. Examination three weeks later, before the patient was transferred to another base, revealed no residual evidence of the original infection.

CASE 2.—This case is typical of 3 cases.

A man aged 26 reported on Dec. 12, 1944, complaining of the typical symptoms of bilateral chronic blepharoconjunctivitis, of eight weeks' duration. Culture revealed the presence of staphylococci. Vision was 20/30 in each eye; with a correction of +0.50 D. cyl., axis 90, it was 20/20, with ability to read Jaeger type 1. As the two eyes were involved equally, it was decided to use the penicillin ointment in the right eye and 5 per cent ophthalmic sulfathiazole ointment in the left eye. Single daily applications for eight days relieved the condition in the right eye, with elimination of swelling and redness of the margins of the lids. Although the left eye felt better, the redness and swelling of the lid margins persisted, and a few scales on the eyelashes were noted. After an interval of one week without treatment, the right eye was still free of infection, while the left eye continued to show a few scales and the presence of infection.

CASE 3.—This case is typical of 5 cases.

A man aged 23 appeared on Dec. 13, 1944 with bilateral chronic blepharoconjunctivitis, of ten years' duration. Vision was 20/20 in each eye, with ability to read Jaeger type 1. In the past he had received treatment with aqueous brilliant green and 5 per cent ophthalmic sulfathiazole ointment, with only partial relief. Treatment with penicillin ointment was instituted; after four days with single daily applications all irritation in both eyes had disappeared, and within two weeks all evidence of the infection was absent. The culture, which was positive for Staph. aureus, became negative within seventy-two hours. Examination two weeks later revealed no evidence of the original infection.

CASE 4.—This case is typical of 2 cases.

A man aged 27 reported on Dec. 20, 1944, complaining of the usual signs and symptoms of bilateral chronic blepharoconjunctivitis. The condition was of three months' duration. Vision was 20/20 in each eye, with ability to read Jaeger type 1. Culture revealed Staph. aureus. With the first application of the penicillin ointment to both eyes, the patient immediately complained of a severe burning sensation, with lacrimation and marked blepharospasm of both eyes. The conjunctiva shortly thereafter appeared greatly congested, with redness of the margins of the lids. Since an allergic reaction was suspected, patch tests were made on both forearms with the penicillin solution, the penicillin ointment and the ointment base. Twenty-four hours later positive reactions were obtained to both the ointment and the penicillin solution. A fresh penicillin ointment was prepared, with care to eliminate any possibility of impurities. The patch test with this freshly prepared ointment gave a negative reaction. Single daily applications of this ointment resulted in complete removal of the infection within twelve days.

CASE 5.—This case is typical of 6 cases.

A man aged 30 reported on Dec. 25, 1944, complaining of bilateral chronic blepharoconjunctivitis, of six weeks' duration. Vision was 20/20 in each eye,

with ability to read Jaeger type 1. Culture revealed Staph. aureus and Str. viridans. Daily single applications of penicillin ointment in the right eye and of 5 per cent sulfathiazole ointment in the left eye, for twelve days, resulted in complete elimination of all signs of the infection in both eyes. Examination two weeks later showed no evidence of the infection in either eye.

Summary.—The data on the 25 cases may be summarized as follows:

Age range	19 to 45 years
Duration of disease	2 to 120 months
Duration of treatment	7 to 20 days
Penicillin and sulfathiazole ointments equally effective	6 cases
Penicillin ointment more effective	17 cases
Reactions	2 cases
No improvement with penicillin ointment	2 cases
Duration of observation	1 to 2 months
Cultures demonstrating Staph. aureus	16 cases
Cultures demonstrating Staph. aureus and Strep. viridans	6 cases
Cultures demonstrating Staph. aureus and nonhemolytic streptococcus	3 cases

COMMENT

Although penicillin in solution is very unstable at room temperatures, its incorporation into an ointment base makes available an ointment which remains potent for four to five weeks at room temperatures, without doubt an effective drug to be added to the armamentarium for ophthalmic therapy. According to the results in this series of 25 cases, the ointment is more effective than drugs formerly used in the treatment of chronic blepharoconjunctivitis due to sensitive staphylococci, with rapid relief of symptoms and clearing of the infection.

Pyle and Rattner [19] and Keyes [16] reported cases in which allergic reactions to penicillin occurred in treatment of ocular infections. In 2 cases what originally was suspected to be an allergic response proved to be due to impurities in the ointment base.

In 6 cases equal improvement was obtained with penicillin ointment and with 5 per cent sulphathiazole ointment. Examination three weeks later showed no evidence of recurrence in the 6 patients treated with the two ointments, the eyes remaining free of infection after cure for at least one month's observation.

In the 2 cases in which no improvement resulted, the question of a noninfectious process, such as allergy, has to be considered.

CONCLUSION

A definitely potent ophthalmic penicillin ointment not requiring refrigeration is available.

Although not a panacea, the ophthalmic penicillin ointment has a decided advantage over drugs formerly used in the treatment of chronic blepharoconjunctivitis.

There was no evidence of an allergic reaction to penicillin in the present series of 25 cases.

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19. Pyle, H. D., and Rattner, H.: Contact Dermatitis from Penicillin, J. A. M. A. **125**:903 (July 29) 1944.

HEREDODEGENERATIVE DISEASES OF THE RETINA

An Attempt at Classification

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ABIOTROPHY

IN A lecture published in the *Lancet* in 1902, Gowers [1] spoke of "death" as having two aspects. There is death of the whole body, somatic death, but there is also death of parts of the body.

> Some of them may slowly die, while the life of all the rest goes on without impairment. They may die from many causes, some early, inevitably, from a grave defect of vital endurance; some much later, the failure being only slightly premature; and some at various times, apparently from various causes. When the failure is early it is often due only to a defect in vitality, a defect which seems to be inherent, the tendency thereto inborn. We do not, indeed, apply the word "death" to this slow decay of the elements; we speak of it as "degeneration," but the process is in many cases, perhaps in most, an essential failure of vitality and I think it is instructive to consider the degenerations in this aspect. But in doing so I am met by the difficulty that we have no word by which to designate this conception—a degeneration or decay in consequence of a defect of vital endurance.[1]

Gowers took the word *biotrophos,* which he found had been used by an ancient writer in the sense of "vital nutrition." By prefixing the negative particle, he coined the word "abiotrophy" for the diseases characterized by "a degeneration or decay in consequence of a defect of vital endurance."

Collins [2] used Cowers' term "abiotrophy" to characterize certain diseases of the eyes, including retinitis pigmentosa and macular degeneration, which, he assumed, were due to a degeneration in consequence of a vital defect.

HEREDODEGENERATION AND HEREDOCONSTITUTIONAL DISEASE

However, many investigators found objections to the term "abiotrophy." The word appeared clumsy, and its etymologic derivation was not clear. According to some, it simply meant premature senility, for which an additional term was not necessary, and it added nothing to the understanding of any disease. Diseases due to a defect in vitality

1. Gowers, W. R.: A Lecture on Abiotrophy, Lancet **1**:1003, 1902.

2. Collins, E. T.: Abiotrophy of the Retinal Neuroepithelium, or "Retinitis Pigmentosa," Tr. Ophth. Soc. U. Kingdom **39**:165, 1919; Hereditary Ocular Degenerations: Ophthalmic Abiotrophies, Tr. Internat. Cong. Ophth. **1**:103, 1922.

attack especially the central nervous system. Jendrassik,[3] who discussed these diseases of the nervous system in a number of publications, spoke of them as hereditary degenerations and created the concept of heredodegeneration. This term, used practically in the same sense in which Gowers used "abiotrophy," seems to be free from some of the connotations which accompany the latter. Behr [4] made use of it in speaking of heredodegeneration of the macula.

Kehrer [5] also preferred "heredodegeneration" to "abiotrophy" and used the term for those hereditary diseases of the nervous system which in the main are characterized by a progressive loss of a specific neural tissue. He expressed agreement with Bielschowsky that under the concept of heredodegeneration should be included conditions in which there is a progressive loss of elements of neural tissue, such as ganglion cells or glia cells. Kehrer also pointed out that in some diseases in which a hereditary vital defect exists there is no progressive loss of tissue and the disease remains stationary for many years and perhaps throughout life. He spoke of these disturbances as "heredoconstitutional" and differentiated them from the progressive "heredodegenerations."

HEREDODEGENERATIONS OF THE RETINA

Among the diseases of the retina are a number which must be classified as heredodegenerations. They have this in common: They are bilateral; they are hereditary and familial; they are not due to any external cause, to inflammation or to vascular changes; with the exception of the heredoconstitutional diseases, they are progressive. Because the elements of the retina are neural or of neural origin, they frequently partake of the heredodegenerative diseases which affect similar elements in the central nervous system. An attempt to classify the heredodegenerative diseases which affect the retina must take into account the fact that the retina is made up of a number of separate elements and that each of these elements is theoretically subject to some form of heredodegeneration. A review of the various diseases of the retina shows that this is actually the case and that the degenerations of the retina can be classified according to the individual layer or kinds of cells primarily affected. The layers of the retina thus subject to some form of heredodegeneration are: 1. The elastic layer of Bruch's membrane. This membrane, although not a part of the retina, is a border membrane

3. Jendrassik, E.: Die hereditären Krankheiten, in Lewandowski, M.: Handbuch der Neurologie, Berlin, Julius Springer, 1911, vol. 2, p. 321.

4. Behr, C.: Heredodegeneration der Makula, Klin. Monatsbl. f. Augenh. **65**:465, 1920.

5. Kehrer, F.: Erbliche organische Nervenkrankheiten: Allgemeine Einleitung, in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1936, vol. 16, p. 222.

forming a barrier between the choroid and the retina. It is subject to degeneration which has consequences for the retina. 2. The pigment epithelium. 3. The neuroepithelium. 4. The nuclear layers. 5. The ganglion cells. 6. The nerve fiber and the glia cells.

Glass Membrane of Bruch.—This membrane consists of two distinct layers: an inner one, which is a product of the pigment epithelium, and an outer elastic layer, which is in immediate touch with the choriocapillaris. The elastic layer forms the termination of the intertwined elastic and collagenous fibrils of the choroid. This layer is subject to degeneration, which shows itself by a change in its staining characteristics and by changes in its width, becoming thickened in some places and thinned in others, to the point of rupture and hole formation. The degeneration causes secondary changes and results in two distinct diseases:

1. Disciform degeneration of the macula (Kuhnt-Junius disease), in which the primary degeneration is limited to the elastic layer of Bruch's membrane.

2. Angioid streaks in the fundus, in which, in addition to the primary degeneration in the elastic layer, there occurs frequently a degeneration of the elastic tissue of some areas of the skin (pseudoxanthoma elasticum) and of the elastic tissue in some blood vessels.

Pigment Epithelium.—I have said that the inner, or basal, layer of Bruch's membrane is a product of the pigment epithelium. In this layer there occur hyaline or colloid bodies, known as such or by the German name of drusen. These hyaline or colloid bodies are of degenerative character. They occur (1) in inflammatory diseases of the eyes; (2) in association with other heredodegenerative diseases of the retina, (3) in advanced age and (4) in young persons. Whenever they occur in the absence of inflammatory diseases, in young or older persons by themselves or in association with other heredodegenerative diseases of the retina, they are the result of a primary vital defect on a hereditory basis of the pigment epithelium.

Neuroepithelium.—The neuroepithelium is funtionally divided into the central, or macular, area and the extramacular, or peripheral, area. Each of these areas is subject to heredodegeneration, which affects them separately or in combination, alone or in association with heredodegenerative diseases of the central nervous system. The neuroepithelium is the seat of typical examples of heredodegeneration, or abiotrophy. Here the retinal elements which have developed begin to disappear at various periods in life.

Central, or Macular, Area.—Degeneration of this area has been described under various names: Hutchinson-Tay choroiditis; familial choroiditis or honeycomb choroiditis; Best's familial congenital

macular degeneration; Stargardt's familial progressive degeneration in the macula and Lutz's hereditary familial chorioretinitis. The multiplicity of names has led to the assumption that the macular area is affected by a number of different degenerative diseases. It is to the credit of Behr that he clarified the situation by showing that all these various familial and hereditary diseases of the macular area constitute one group—heredodegeneration of the macula. He found that (1) all these conditions present a uniform and characteristic picture; (2) that underlying the clinical picture is a familial and hereditary degeneration of the macula; (3) that the age at which the disease manifests itself varies in the different families and that it usually occurs at the age of 6 to 8 years (infantile type), of 12 to 14 years (juvenile type), in the twenties (adult type), in the late forties and early fifties (presenile type) and still later (senile type) and (4) that the disease appears either alone or in combination with other diseases of the retina or of the central nervous system.

Hereditary degeneration of the macular area can therefore occur (1) alone or in association with (2) degeneration of the peripheral areas of the retina, (3) atrophy of the optic nerve, (4) color blindness or (5) mental deterioration.

These combinations do not constitute separate forms of macular degeneration. In them there are distinct hereditary diseases which occur in association, a not uncommon occurrence in the central nervous system.

Extramacular Neuroepithelium.—The typical heredodegenerative disease of the extramacular neuroepithelium is retinitis pigmentosa. The disappearance of the rods and cones begins in the region of the equator and proceeds centrally. It is the disease which Collins used to illustrate abiotrophy of the retinal elements. Retinitis pigmentosa is inherited in three modes: (1) as a recessive, the most frequent mode in heredity; (2) as a dominant, which is much less frequent, and (3) as a sex-linked recessive, which is rare. The disease occurs alone or in association with other forms of heredodegeneration: (1) degeneration of the macular area; (2) color blindness; (3) deafness; (4) mental deterioration; (5) the juvenile type of amaurotic familial idiocy, and (6) the Laurence-Moon-Biedl syndrome, in association with obesity, polydactyly, hypogenitalism and mental deterioration.

In addition to heredodegeneration, the neuroepithelium is also subject to heredoconstitutional diseases in the sense of Kehrer. These diseases are hereditary and familial but remain practically stationary throughout life. They are:

1. Retinitis puntata albescens, in which the night blindness is practically stationary or very slowly progressive. The form of heredity is probably recessive.

2. Congenital night blindness without changes in the fundus. The condition remains unchanged throughout life. It is inherited in three forms: as a dominant (the Nougaret type), as a simple recessive and as a sex-linked recessive.

3. Oguchi's disease (congenital night blindness with grayish white discoloration of the fundus). The disease is familial, and its form of heredity is probably recessive. It remains stationary throughout life.

4. Color blindness.

In addition to these heredodegenerations and heredoconstitutional diseases of the neuroepithelium, there are two diseases of unknown origin which stand in some relation to them:

1. Gyrate atrophy of the choroid and retina. There is no doubt that this disease is a familial heredodegeneration involving both the retina and the choroid. Which is the primary membrane involved is not known.

2. Choroideremia. This is probably a heredoconstitutional disease and has been observed in brothers. It is stationary throughout life, and other forms of heredodegeneration have been found in members of the families of reported patients. Mann stated the opinion that the sporadic cases of choroideremia are possibly purely developmental, the choriocapillaris having failed to form.

Ganglion Cells of the Retina.—Heredodegeneration of the ganglion cells of the retina occurs in three distinct diseases, which also affect the ganglion cells in all parts of the central nervous system:

1. The infantile type of amaurotic familial idiocy (Tay-Sachs disease).

2. The juvenile type of amaurotic familial idiocy (Vogt-Spielmeyer or Stock-Spielmeyer or Batten-Mayou type). Included also is the late infantile type, which is, strictly speaking, an early juvenile type.

3. Niemann-Pick disease.

The three diseases have in common a pathologic process which involves primarily the ganglion cells of the central nervous system, including those of the retina. The essential change is an accumulation of prelipids (sphingomyelin) in the ganglion cells, with eventual degeneration of the cells. The diseases are familial and hereditary, and it is possible that they are primarily heredoconstitutional instead of heredodegenerative.

They differ among themselves as follows:

1. In Tay-Sachs disease the ganglion cells of the central nervous system, including those of the retina, are affected.

2. In Niemann-Pick disease there is, in addition, an accumulation of foam cells filled with lipids in the liver, spleen, lymph nodes, thymus, adrenal glands, kidneys, stomach, intestine, pancreas and heart muscle.

3. In the juvenile type of amaurotic familial idiocy there is, in addition to the changes in the ganglion cells of the retina and of the central nervous system, an associated degeneration of the extramacular neuroepithelium (retinitis pigmentosa).

Nuclear Layers.—Degeneration of the nuclear layers, especially of the inner layer, occurs as a primary degeneration in the following conditions:

1. As peripheral cystoid degeneration in senile, presenile and myopic eyes in the anterior portion of the retina near the ora serrata. It extends backward about 7 mm., rarely more. Its occurrence in presenile and senile eyes speaks for degeneration on a hereditary basis. In some eyes the cystoid degeneration is extensive and leads to rupture of the retina, to hole formation and to consequent retinal detachment.

2. Retinitis circinata. Only a small number of cases of this condition have been studied histologically. In these a cystoid degeneration involved the nuclear and internuclear layers, with deposits of hyalin and fat and the presence of fat granule cells. The cystoid degeneration was particularly pronounced in the central area. The disease is not a common one and heredity studies are not available. However, such a disappearance of retinal elements, resulting in the formation of cystoid spaces, clearly indicates a primary degeneration and can only occur as a result of a hereditary defect.

Nerve Fiber Layers of the Retina.—There is no known heredodegeneration of the nerve fibers of the retina. But there is a disease of hereditary character affecting the central nervous system which also involves the nerve fiber layer of the retina, namely, tuberous sclerosis. This disease is difficult to classify. It is a heredodegenerative or heredoconstitutional disease and is also a congenital anomaly. It is a system disease in that it involves tissues over widespread areas in the body. Some investigators emphasize the neoplastic tendency of the tissues; others consider it the result of an early embryonic disturbance in organ formation. In the retina there occur in this disease tumors which seem to be derived from the nerve fiber layer and from glia cells in this and in the ganglion cell layers.

SUMMARY

Under "heredodegeneration" are classified the diseases of the central nervous system which in the main are characterized by a specific loss of neural tissue as a result of hereditary influences. The term "abiotrophy" has essentially the same significance. By "heredo-

constitutional disease" is understood a disease which is the result of a hereditary vital defect in some tissue but in which there is no progressive loss of the tissue and the disease remains stationary for many years or throughout life.

Such diseases occur in the retina and are found here under the following conditions:

1. As a primary, separate and distinct disease of certain retinal elements. Examples are retinitis pigmentosa and heredodegeneration of the macula.

2. In association with another heredodegenerative disease of the retina. An example is degeneration of the macula associated with degeneration of the neuroepithelium in the periphery.

3. In association with hereditary diseases of the central nervous system. Examples are degeneration of the macula associated with mental deterioration and retinitis pigmentosa in the Laurence-Moon-Biedl syndrome.

4. As a part of a hereditary disease of the central nervous system. An example is amaurotic familial idiocy.

5. As a part of a widespread system disease involving many organs. Examples are Niemann-Pick disease and tuberous sclerosis.

The individual heredodegenerative diseases of the retina involve primarily certain retinal elements and can be classified as follows:

A. Originating in the elastic layer of Bruch's membrane
   1. Disciform degeneration of the macula (Kuhnt-Junius type)
   2. Angioid streaks in the fundus
      (*a*) Occurring alone
      (*b*) Occurring in association with pseudoxanthoma elasticum (Grönblad-Strandberg syndrome)

B. Originating in the pigment epithelium
   1. Hyaline or colloid bodies (drusen) of the basal layer of Bruch's membrane

C. Originating in the neuroepithelium
   I. In the central, or macular, area
      1. Heredodegeneration of the macula, including the infantile, juvenile, adult, presenile and senile types, occurring (*a*) alone or in association with (*b*) degeneration of the peripheral neuroepithelium, (*c*) atrophy of the optic nerve, (*d*) color blindness and (*e*) mental deterioration
   II. In the extramacular neuroepithelium
      1. Retinitis pigmentosa, occurring (*a*) alone, or in association with (*b*) degeneration of the macula, (*c*) color blind-

ness, (*d*) deafness, (*e*) mental deterioration, (*f*) the juvenile type of amaurotic family idiocy and (*g*) the Laurence-Moon-Biedl syndrome.

III. Heredoconstitutional diseases of the neuroepithelium

1. Retinitis punctata albescens
2. Congenital night blindness without changes in the fundus
3. Congenital night blindness with grayish white discoloration of the fundus (Oguchi's disease)
4. Color blindness

IV. Heredodegeneration of doubtful origin but standing in some relation to degeneration of the neuroepithelium

1. Gyrate atrophy of the choroid and retina
2. Choroideremia, a heredoconstitutional disease

D. Originating in the ganglion cells

1. Infantile type of amaurotic familial idiocy (Tay-Sachs disease), affecting only the ganglion cells
2. Juvenile type of amaurotic familial idiocy, involving the neuroepithelium in addition to the ganglion cells
3. Niemann-Pick disease

E. Involving the nuclear layers

1. Peripheral cystoid degeneration in senile and presenile eyes
2. Widespread cystoid degeneration, forming the basis for rupture of the retina with hole formation and consequent retinal detachment
3. Retinitis circinata

F. Involving the nerve fiber layer

1. Tuberous sclerosis, a widespread heredodegenerative disease, causing the production of abnormal cells and tumors in this layer

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ACTION SPECTRUM OF KERATITIS PRODUCED BY ULTRAVIOLET RADIATION

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ACKNOWLEDGE of the distribution of wavelengths and energies involved in the production of keratitis by ultraviolet radiation has theoretic and practical importance. Such information is fundamental to an understanding of the harmful photobiologic processes in the cornea and is prerequisite to the determination of exposure hazards and protective devices. The present report attempts to show quantitatively what bands of the ultraviolet portion of the spectrum are responsible for the production of keratitis and what relation the keratitis band has to the erythema band, to the characteristic absorption bands of some proteins and to the absorption of several types of common glass.

A curve relating wavelength to the quantity of energy just necessary to produce keratitis represents the action spectrum for keratitis, whereas a curve relating wavelength to the absorption of the corneal epithelium represents the absorption spectrum of the corneal epithelium. It is one aim of this study to correlate the action spectrum of keratitis with the absorption spectrum of a particular tissue constituent.[1]

The action spectrum for keratitis has not previously been determined, although the long wavelength limit which would produce keratitis has been determined by use of filters, as in the standard work of Verhoeff and Bell.[2]

PROCEDURE AND APPARATUS

The eyes of rabbits (albino) were exposed to separate bands of ultraviolet radiation at energy levels having a sufficient spread to determine the threshold dose necessary to produce keratitis.[3]

This study was supported in part by a grant made to the Howe Laboratory of Ophthalmology by the American Optical Company.

From the Howe Laboratory of Ophthalmology, Harvard Medical School, and the Massachussetts Eye and Ear Infirmary.

1. A subsequent report by one of us (V. E. K.[8]) will show the characteristic absorption of various parts of the eye for the ultraviolet portion of the spectrum. The data in that paper will be drawn on freely where needed in the present report.

2. Verhoeff, F. H., and Bell, L.: The Pathological Effects of Radiant Energy on the Eye, Proc. Am. Acad. Arts & Sciences **5**:629-818, 1916.

The apparatus used was a Bausch and Lomb double monochromator (type no. 33-86-05) containing two 8 by 8 cm. quartz prisms, two quartz lenses and a high pressure General Electric mercury vapor lamp of 1,000 watt capacity.[4]

The quantity of radiant energy was determined each day of experiment by means of a thermopile. This had been calibrated in absolute units of energy by exposing it to radiations emanating from a small opening of a furnace constructed in such a way as to possess all the characteristics of a black body radiator. The radiant flux measured in this manner is expressed in ergs per minute per square centimeter, from which the radiant energy, expressed in ergs per square centimeter, incident on the cornea is given by multiplying the radiant flux by the period of radiation employed. The measurements of energy are believed to have been determined with an accuracy of ± 25 per cent at the short end of the spectrum used and of ± 10 per cent at the long end.

The quality of the radiations was calculated from data supplied by Slaunwhite,[5] in whose thesis is a detailed description of the method employed. All the exposures were carried out with entrance and exit slits set at 1 mm. With

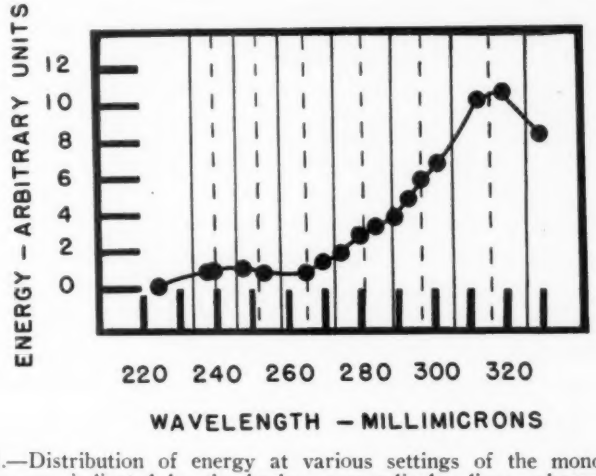

Chart 1.—Distribution of energy at various settings of the monochromator. The settings are indicated by the broken perpendicular lines, whereas the total wave band for each setting is indicated by the blocked perpendicular lines.

these slit widths, the spectral region from 240 to 316 millimicrons was covered by six different bands approximately 20 millimicrons wide. To determine the peak sensitivity, two other bands, 280 to 296 millimicrons and 296 to 314 millimicrons, were used with the monochromator set at 288 and 305 millimicrons, respectively. With these two exceptions, the bands used are indicated by the

3. The energy necessary to produce keratitis with ultraviolet radiation in man has previously been determined to be approximately two-thirds that necessary in rabbits (Kinsey, V. E.; Cogan, D. G., and Drinker, P.: Measuring Eye Flash from Arc Welding, J. A. M. A. **123**:403-404 [Oct. 16] 1943).

4. Loaned by Dr. J. R. Loofborrow and Prof. Francis Schmitt, of the Department of Biology at the Massachusetts Institute of Technology.

5. Slaunwhite, W. R. Jr.: An Investigation of the Rise of Monochromatic Ultraviolet Radiation in the Preparation of Vaccines, Thesis, Massachusetts Institute of Technology, Department of Biology and Public Health, 1942.

blocked perpendicular lines in figure 1, and the monochromator settings, by broken lines.

The absorption measurements for the glass filters and protein solutions were made with the Beckman spectrophotometer.[6] The nuclear protein used was prepared by extracting corneal epithelium with water and precipitating with 33 per cent ammonium sulfate. The serum used was simply diluted with water.

The dose was varied by controlling the duration of exposure, it being well established that these photobiologic processes obey the law of reciprocity.[7]

During the exposure the rabbits were held immobile in a box, with head protruding through the end. The lids were held apart manually and the eyes exposed for the desired length of time with the beam incident on the center of the cornea. General anesthesia (pentobarbital) was rarely necessary, and then only with the extremely long exposures used in the experiments in which glass filters were interposed between the exit slit and the eye of the rabbit.

The eyes were examined with the biomicroscope and slit lamp before the exposure and at successive intervals after the exposure of one day (eighteen hours) and two, four and seven days, or until the corneas had returned to normal.

A total of 137 rabbit eyes were exposed. Of these, the data on 9 are not recorded because of the development of corneal abrasions or other abnormalities which vitiated their inclusion in the series.

The threshold dose was arrived at not only by determining the energy just necessary to produce an ocular reaction but by determining the transition between consistently negative and consistently positive results with increasing doses. With rare exceptions, these two criteria were in agreement.

GRADING OF OCULAR REACTION

The most reliable sign of keratitis with threshold reactions was, in our opinion, a granular appearance within the corneal epithelium seen by transillumination with the slit lamp and biomicroscope. The granules were of uniform size, each granule being of the order of size of a single epithelial cell. The individual granules were round, but because of their small size no further details could be seen with a magnification of 40. With threshold reactions there were 50 to 200 of the individual granules, while with more severe reactions there was a corresponding increase in the number of granules, ultimately forming a mosaic. Because of their size and uniformity and because of the occurrence of similar granules after irrigation with tap water, the granules are presumed to be swollen epithelial cells. Histologic examination of corneas showing these minimal ocular reactions showed, with the usual fixatives and stains, nothing unusual.

Associated with this granular appearance were a stippling of the surface of the cornea, seen in reflected light, and a varying amount of fluorescein "take." However, stippling is more difficult to assess quanti-

6. Loaned by Dr. Theodore L. Terry, of the Department of Pathology of the Massachusetts Eye and Ear Infirmary.

7. Coblentz, W. W.; Stair, R., and Hogue, J. M.: The Spectral Erythemic Reaction of the Untanned Human Skin to Ultra-Violet Light Radiation, Bureau of Standards J. Research **8**:541-547, 1932.

tatively, and fluorescein "take" shows such a wide variation among normal rabbit eyes as to be misleading in the detection of minimal changes. Redness and discharge were even less reliable indexes. The criteria for evaluating the threshold reaction were therefore weighted heavily in favor of the granules.

With relatively severe exposures the corneal stroma became hazy. It is of interest that the cloudiness of the stroma occurred only with amounts of radiation which would be expected on the basis of transmission measurements [8] to penetrate the cornea in sufficient quantities to produce a threshold reaction in the endothelium.

The ocular signs were maximal within the first two days after exposure, but their duration afforded a check on the reliability of the initial appearance. Only with severe exposures were residual signs to be found after seven days. During this time the granules disappeared, leaving an apparently normal cornea, in the case of threshold exposures, or one with varying degrees of optical irregularity and epithelial edema, in the case of suprathreshold exposures.

For tabular purposes, the ocular reaction was graded — to ++++, according to the following arbitrary differentiation. Thus — indicated no abnormality; ± indicated a probable but not definite abnormality; + indicated 50 to 200 granules with return to normal by the fourth day; ++ indicated massive granules in the cornea for the first two days, optical irregularity on the fourth day and a return to normal by the seventh day with no cloudiness of the cornea at any time; +++ indicated massive granules during the first two days with mild cloudiness of the stroma, usually clearing by the seventh day, and ++++ indicated massive granular appearance during the first two days, considerable opacification of the stroma, loss of much of the epithelium and definite abnormalities still evident on and after the seventh day.

RESULTS

The ocular reactions produced by different energies at various wavelengths are represented in figure 2, with blocking of what we consider to be the threshold reactions. These threshold reactions are plotted in figure 3 to show the action spectrum or sensitivity at various wavelengths.

The significant features of the action spectrum are the shape of the curve, the point of maximal sensitivity and the long wavelength limit of reaction. The curve is obviously abrupt, with a peak at 288 millimicrons, and shows by extrapolation that the long wavelength limit lies somewhere between 306 and 326 millimicrons.

8. Kinsey, V. E.: Spectral Transmission of the Eye to Ultraviolet Radiation, to be published.

Figure 3 represents the action spectrum for keratitis, the action spectrum for erythema (Coblentz, Stair and Hogue [7]) and the characteristic absorption spectrums of serum and nucleoprotein. The shapes of the

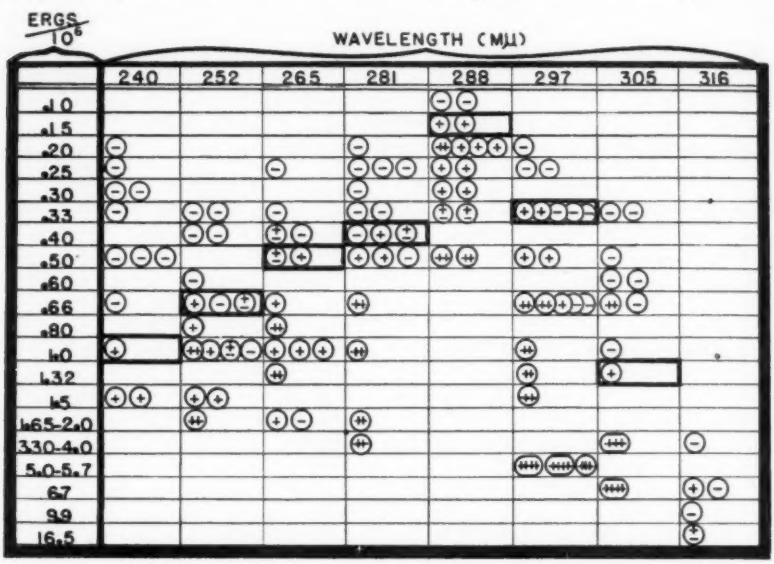

Chart 2.—Reactions in rabbit corneas produced by exposure to bands of ultraviolet radiation of various wavelength at different energy levels. What are interpreted as threshold reactions are indicated by blocking.

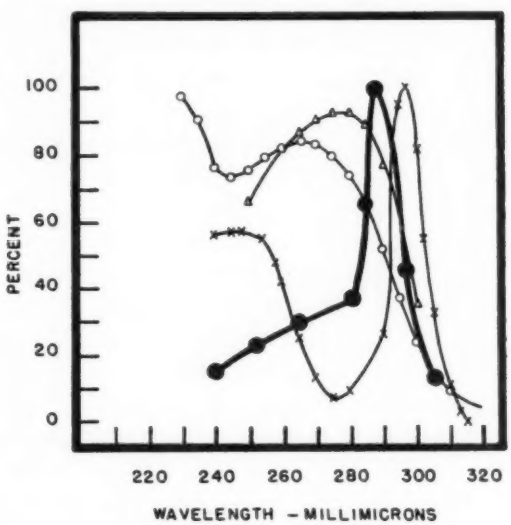

Chart 3.—Curves illustrating the sensitivity of the cornea (line and solid dots) and of the skin (line and crosses) to bands of different wavelength and the characteristic absorption of nucleoprotein (line and hallow circles) and of serum protein (line and triangles)

keratitis and the erythema curve are similar; their difference lies chiefly in their maximums. The erythema peak is shifted more to the right than is the keratitis peak, and both are somewhat to the right of the nucleoprotein peak. The keratitis peak most nearly coincides with the serum peak. The significance of these differences will be discussed subsequently.

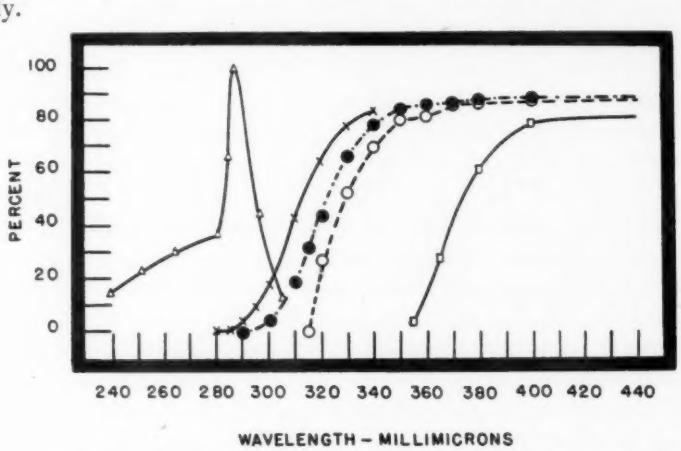

Chart 4.—Curves illustrating the action spectrum of keratitis (line and triangles) and the absorption by glass of 2 mm. thickneess of the following types: crown or spectacle glass (line and crosses), window glass (line and dark circles), plate glass (line and hollow circles) and flint glass (line and rectangles).

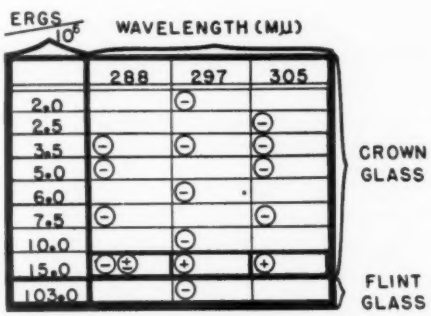

Chart 5.—Reactions in rabbit corneas produced by exposure to bands of various wavelength when glass (2 mm. thick) is interposed between the source of energy and the eye. The energies indicated are those which would be incident on the eye if no glass were used.

The energy necessary to elicit a threshold reaction in the cornea at the peak of sensitivity was of the order of 0.15 by 10^6 ergs. This is to be compared with the value of 2.0 by 10^6 ergs previously found[9] when the whole ultraviolet portion of the spectrum was utilized.

9. Verhoeff and Bell.[2] Duke-Elder, W. S.: Pathologic Action of Light upon the Eyes, Lancet **1**:1137-1140 (June 12) 1926.

The relation of the action spectrum of keratitis to the transmission of various types of common glass is represented in figure 4, and some experiments on rabbit eyes, using glass filters of 2 mm. thickness, are presented in figure 5. In these experiments, the various types of glass were interposed between the exit slit of the monochromator and the rabbit eye. From figures 4 and 5 it is apparent that a 2 mm. thickness of crown glass reduces the exposure hazard approximately fifteenfold at 305 millimicrons, forty-five fold at 297 millimicrons and a hundred fold at 268 millimicrons. A 2 mm. thickness of flint glass affords essentially complete protection at all wavelengths.

COMMENT

The photosensitive substance in skin responsible for erythema after ultraviolet irradiation has been assumed to be protein in nature, and the lack of correspondence between the action spectrum of erythema and the absorption curve of the skin has been assumed to be due to the absorption and differential scattering effects of the superficial, inert layer of the skin. Thus, the interposition of a solution of "typical protein" in the path of the radiation results in a shift in energy transmitted, with a resultant curve similar to the erythema curve.[10] And, conversely, diminishing the scattering effect of the superficial layer by suitable clearing reagents results in an absorption curve for the whole epidermis similar to that for protein (serum albumin).[11] Presumably, therefore, if it were possible to eliminate the effect of the superficial, inert layer of the skin, the erythema curve would be shifted toward the shorter wavelengths, to have a peak sensitivity similar to that of protein at about 280 millimicrons. In the case of the skin, therefore, there is a reasonably good correlation between absorption by the epidermis and the erythema reaction.

The cornea has no superficial layer corresponding to that in the skin. It is interesting, therefore, to find that the peak of the action spectrum for keratitis corresponds approximately to that of the "corrected" erythema spectrum. But whereas the corrected erythema spectrum corresponded approximately to the corrected absorption curve for skin, both having peaks at about 280 millimicrons, the keratitis peak does not correspond to the absorption peak of the corneal epithelium; the keratitis peak is at 288 millimicrons, whereas the absorption peak of the corneal epithelium is at 265 millimicrons. Thus, while the values for the erythema spectrum are compatible with the assump-

10. Mitchell, J. S.: The Origin of the Erythema Curve and the Pharmacological Action of Ultra-Violet Radiation, Proc. Roy. Soc., London, s.B **126**:241-261 (Oct.) 1938.

11. Lucas, N. S.: The Permeability of Human Epidermis to Ultra-Violet Irradiation, Biochem. J. **25**:57-70, 1931.

tion that the ultraviolet radiation causes a general protein denaturation in the skin, since absorption and effect coincide, the values for the cornea indicate a selective effect at wavelengths other than those which are absorbed maximally by the cornea. The fact that the maximum absorption of the cornea occurs at 265 millimicrons suggests that nucleoprotein is chiefly responsible for the absorption, whereas keratitis occurs with a maximum that more nearly corresponds to that of the cytoplasmic proteins albumin and globulin.

From the absorption curve of corneal epithelium, there is no evidence of selective absorption at wavelengths corresponding to the peak of the action spectrum causing keratitis. This suggests that the amount of photosensitive substance present (albumin, globulin or other substance) is small, e. g., an enzyme, or, alternatively, that the abiotic effects are due to absorption by some small fraction of the protein complex having absorption maximums similar to the keratitis maximum.

SUMMARY

The action spectrum for keratitis produced by radiation was determined on rabbit eyes. Relatively homogeneous radiations with sufficient energy to produce keratitis were obtained by means of a large quartz monochromator. The amount of energy at any one wavelength just necessary to produce a corneal change that was visible with the biomicroscope and slit lamp was considered the threshold dose for that wavelength.

The cornea was found to have a peak sensitivity to ultraviolet radiations at wavelengths of about 288 millimicrons, with a sharp decline in sensitivity to either side of the peak. The amount of energy necessary to elicit an ocular reaction at 288 millimicrons was approximately 0.15×10^6 ergs per square centimeter. Although the absorption peak of the corneal epithelium corresponded to that of nuclear protein (265 millimicrons), the peak of the action spectrum corresponded more nearly to the absorption peak of albumin and globulin (280 millimicrons). From this it may be inferred that the photochemical reaction in the cornea is due not to an indiscriminate absorption by nucleoprotein but, rather, to a selective absorption by a substance having a peak in the wavelengths longer than that of nucleoprotein or by certain constituents only of the nucleoprotein molecule.

The transmission characteristics of various types of common glass were measured for that portion of the spectrum which is responsible for keratitis. By comparison of these characteristics with the action spectrum for keratitis, it is possible to determine the amount of protection provided by any one glass for any wavelength or group of wavelengths.

243 Charles Street (14).

INTRAORBITAL MELANOSIS AND INTRACRANIAL NEUROEPITHELIOMA OF THE OPTIC NERVE

Report of a Case

CHARLES A. PERERA, M.D.
AND
ARTHUR PURDY STOUT, M.D.
NEW YORK

H. A. N., a 21 month old, irritable Armenian girl, was admitted to the service of Dr. A. B. Reese in the Institute of Ophthalmology in November 1943, with the history of an inflamed right eye for one month. The past history was irrelevant except for chickenpox at the age of 7 months and rubella at the age of 16 months. Examination, with the patient under ether anesthesia, showed a normal left eye. The right eye was enlarged and congested, with clear media, increased intraocular pressure (60 mm. Schiøtz) and extremely deep cupping of the optic nerve head (bottom of excavation seen with —15 D. of the ophthalmoscope). In January 1944 iridencleisis with anterior sclerectomy was performed. Although the intraocular pressure became normal, the surgical procedure was followed by recurring intraocular hemorrhages, the development of a grayish white mass behind the lens and the loss of the reddish fundus reflex. The child acquired measles, and the right eye became more irritable. Enucleation of this eye was performed on June 2, 1944 by Dr. James McGraw. On removal of the globe, inspection of its posterior portion showed a ring of pigmented tissue around the entrance of the optic nerve. The orbit revealed an extension of this friable black tissue, which was dissected out. It seemed to extend about 1 inch (2.5 cm.) along the optic nerve posterior to the globe. A gold sphere was implanted into Tenon's capsule. Healing took place uneventfully.

On Nov. 22, 1944 the child was admitted to Babies Hospital in coma, after four days of vomiting. Physical examination showed elevation of the blood pressure (138 systolic and 110 diastolic), papilledema of the left eye, absence of the pupillary reflex, immobility of the left eyeball, trismus, absence of reflexes and flaccidity of all muscles of the extremities. Roentgenograms of the skull showed widened sagittal and coronal sutures, indicative of increased intracranial pressure. Blood cultures yielded no growth. A diagnosis of intracranial neoplasm was made. Supportive treatment was of no avail, and the patient died on November 23. The temperature, which was 100.6 F. on her admission, rose to 105.6 F. just before death. An autopsy was performed by Dr. Beryl Paige, who found no essential abnormalities except a tumor at the base of the brain.

This case was presented before the New York Academy of Medicine, Section of Ophthalmology, Jan. 15, 1945. An abstract of the paper was published in the April 1945 issue of the ARCHIVES, page 329.

From the Department of Pathology of the Institute of Ophthalmology of Presbyterian Hospital, and the Departments of Ophthalmology and Surgical Pathology of Columbia University College of Physicians and Surgeons.

REPORT OF PATHOLOGIC STUDIES

Right Eyeball (described by one of us [C. A. P.]).—Superiorly, the corneosclera showed an interruption of its continuity, and through the interruption extended pigmented, disorganized and atrophic iris tissue. The cornea superiorly revealed a dense fibrous membrane on its posterior surface, extending 2 mm. from the limbus and reaching the curled-up end of Descemet's membrane. The anterior chamber was almost completely abolished except for a small portion inferiorly, the lens being pressed against the cornea. The ciliary body and the iris showed extensive degenerative changes, with dispersal of pigment. Extending

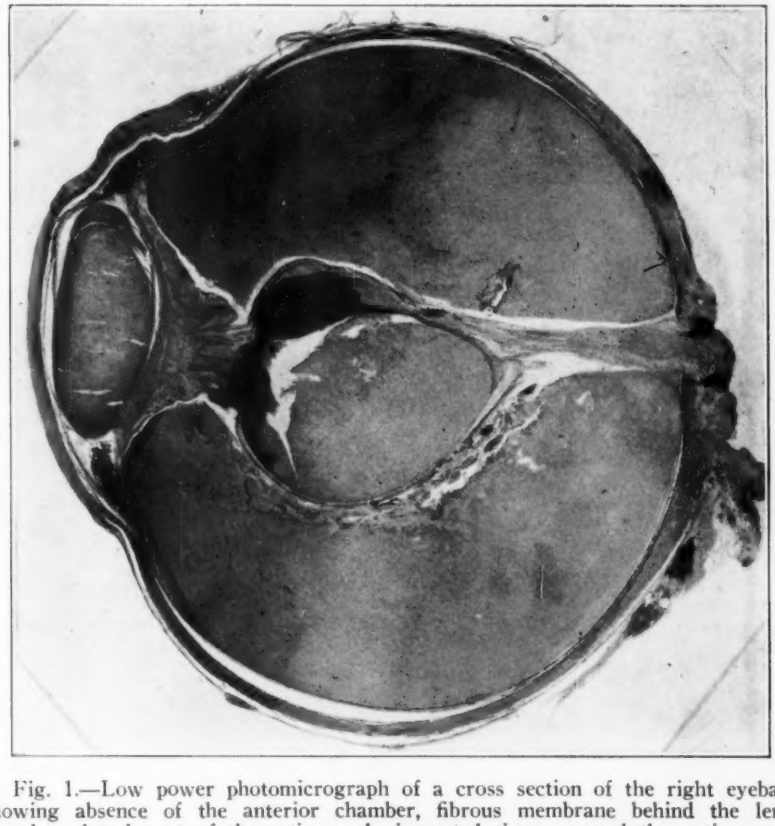

Fig. 1.—Low power photomicrograph of a cross section of the right eyeball, showing absence of the anterior chamber, fibrous membrane behind the lens, complete detachment of the retina and pigmented tissue around the optic nerve.

across the eye behind the lens was a dense connective tissue membrane, to which the lens was adherent and into which was pulled the completely detached retina (fig. 1). Immediately behind this fibrous membrane was a moderate amount of hemosiderin engulfed in phagocytes, as well as several large hemorrhages in the remains of the vitreous cavity. The subretinal fluid showed hemorrhage in its anterior portion. The choroid presented a moderate degree of pigmentation, and the lamina fusca contained a considerable number of chromatophores. At the posterior portion of the globe, the optic nerve had been pulled into the

interior of the eyeball by the dense retrolental membrane and the detached retina. The peripheral limits of the optic nerve sheath contained large numbers of chromatophores, which extended anteriorly to the level of the sclera (figs. 2 and 3). Among the pigmented cells behind the globe were seen rounded hyaline bodies. Stains made with the Perl and the Turnbull method showed that the pigment in the optic nerve was not derived from blood pigment.

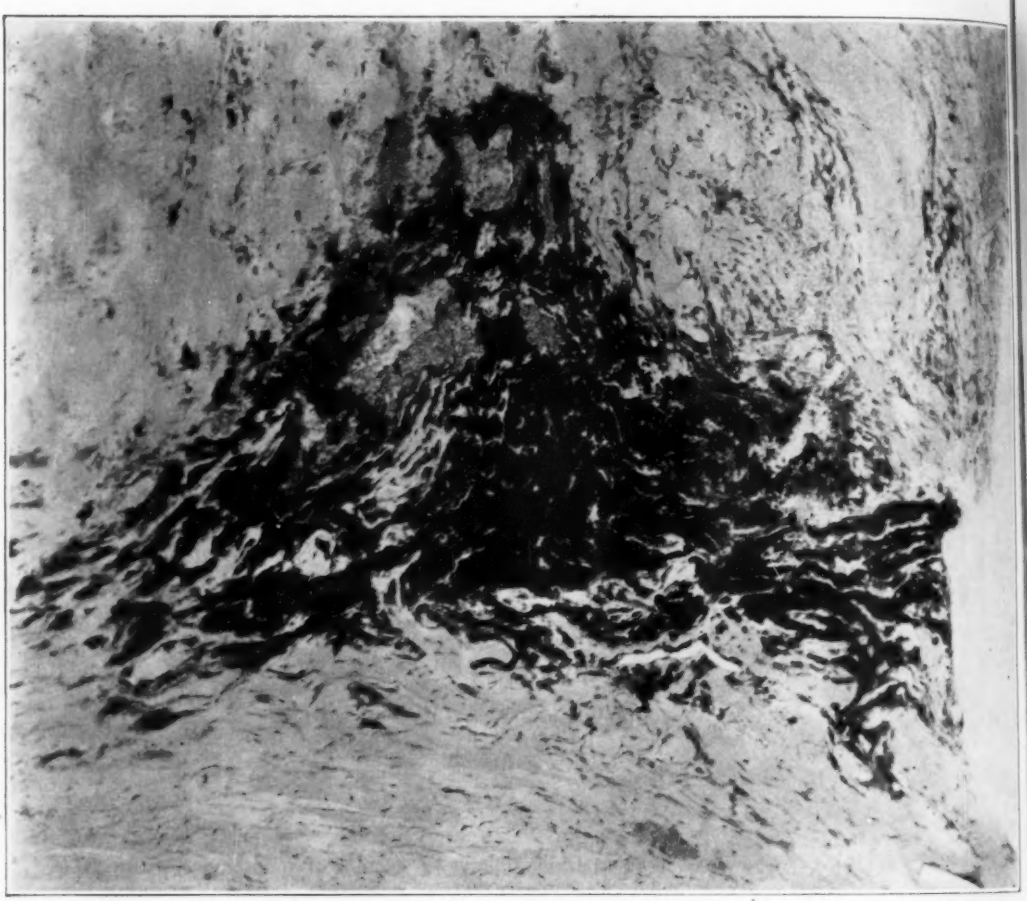

Fig. 2.—Detail of melanotic tissue bordering the optic nerve adjacent to the sclera.

Tissue from Right Orbit (described by one of us [C. A. P.]).—The sections consisted of several small fragments of tissue from the orbit behind the globe. The significant features of these pieces were the extensive atrophy and gliosis of the optic nerve and the invasion of the periphery of this degenerated nerve tissue by pigment-containing cells, which formed an irregular layer around the optic nerve (fig. 4). Internal to this layer of pigmented cells and interspersed

among them were seen many pale-staining, hyalin-like, rounded masses, some of which contained a small, endothelium-lined central lumen (fig. 5). There were also a number of small, rounded, calcified concentrically laminated bodies (fig. 6). The Perl and Turnbull stains showed that the brown pigment in these sections was not derived from blood. Specific stains revealed that the rounded, hyaline

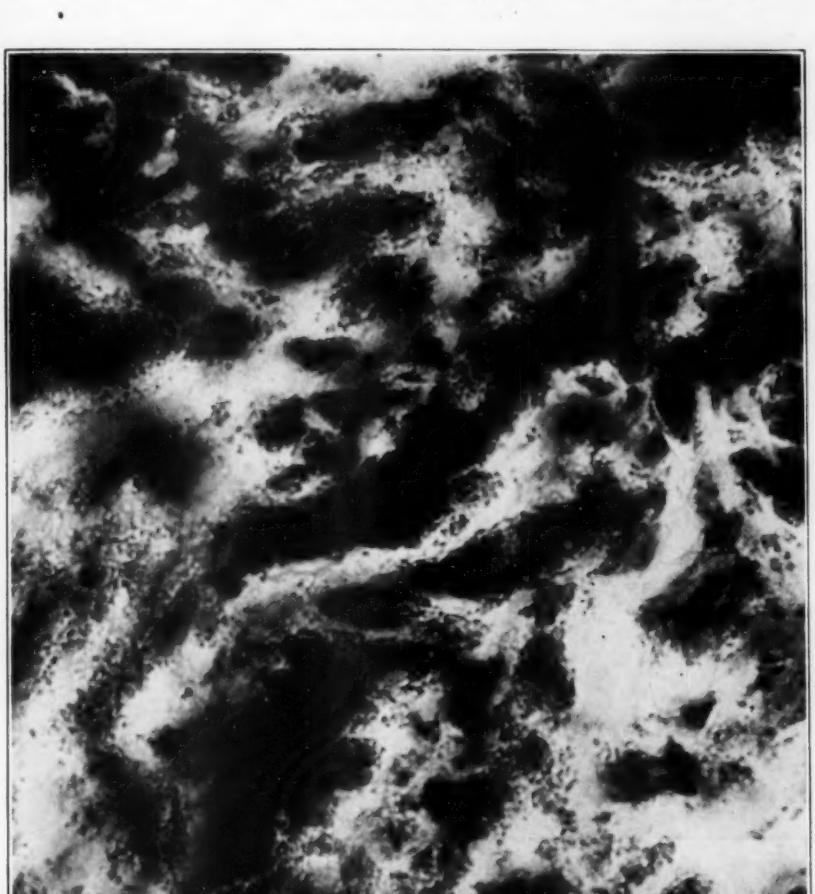

Fig. 3.—High power study of pigment-containing cells shown in figure 2.

masses did not contain elastic tissue. The degenerated optic nerve had a dense external dural coat.

Conclusions from Pathologic Studies of the Eye.—The globe was the seat of a previous glaucoma, for which iridencleisis was performed. Subsequent to this operation there developed intraocular hemorrhages, with the formation of a dense connective tissue membrane behind the lens and complete detachment of the

retina. In the optic nerve behind the globe was seen an unusual pigmented lesion, associated with rounded, hyaline masses and small psammoma bodies. It appeared that the lesion of the optic nerve involved the arachnoid and pial layers of its sheath and that there was a melanosis associated with hyaline degeneration of the blood vessels of the arachnoid layer.

The pathologic diagnosis was hydrophthalmos, detachment of the retina following hemorrhage and contracture, melanosis of the optic nerve, iris inclusion operation.

Brain and Optic Nerves (described by Dr. Abner Wolf).—The cerebral hemispheres were approximately symmetric. They were enlarged and showed pronounced flattening of their gyri. In the midline at the base of the brain a tumor was found, diffusely infiltrating the hypothalamus, the optic chiasm, the lamina

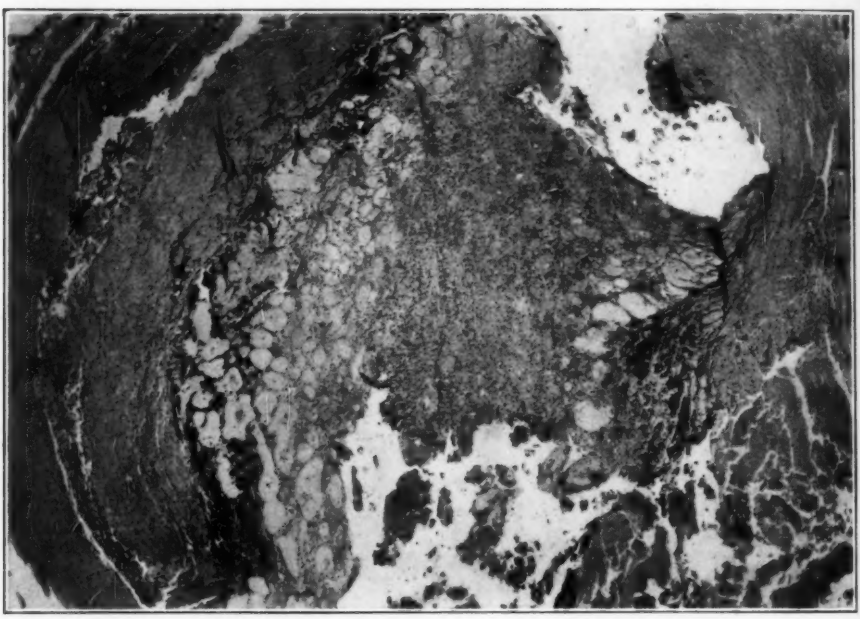

Fig. 4.—Low power photomicrograph of a cross section of the orbital portion of the right optic nerve, showing atrophic nerve fibers, surrounded by rounded hyaline bodies, pigmented cells and a dense dural sheath.

terminalis and the anterior extremity of the right optic tract. The right optic nerve was not separately visualized but was lost in the neoplasm. The left optic nerve was slightly swollen at its origin from the optic chiasm but appeared relatively normal anterior to that point. The tumor-infiltrated lamina terminalis was opaque and bulged forward as a large sac, approximately 1.7 cm. in diameter, which compressed the posterior extremities of the gyri recti, the olfactory trigone, and the posterior third of the right olfactory tract. It extended asymmetrically to the right and elevated the anterior cerebral arteries. The tumor ended in the midhypothalamus. The remainder of the hypothalamus bulged and appeared somewhat hemorrhagic. On midsagittal section of the brain, the neoplasm described externally was found to extend through the hypothalamus into the

anterior two fifths of the third ventricle, partially obliterating it. It extended backward along the right lateral wall of the ventricle to the massa intermedia. The left lateral wall was the site of petechial hemorrhages. It appeared free from tumor. The rest of the ventricle was filled by a recent blood clot, with hemorrhagic tumor anteriorly. This had considerably dilated the cavity and obstructed the much enlarged interventricular foramens. The lateral ventricles were considerably dilated. The cerebellum and the brain stem showed no external abnormalities except for flattening of the dorsal surface of the cerebellum. The

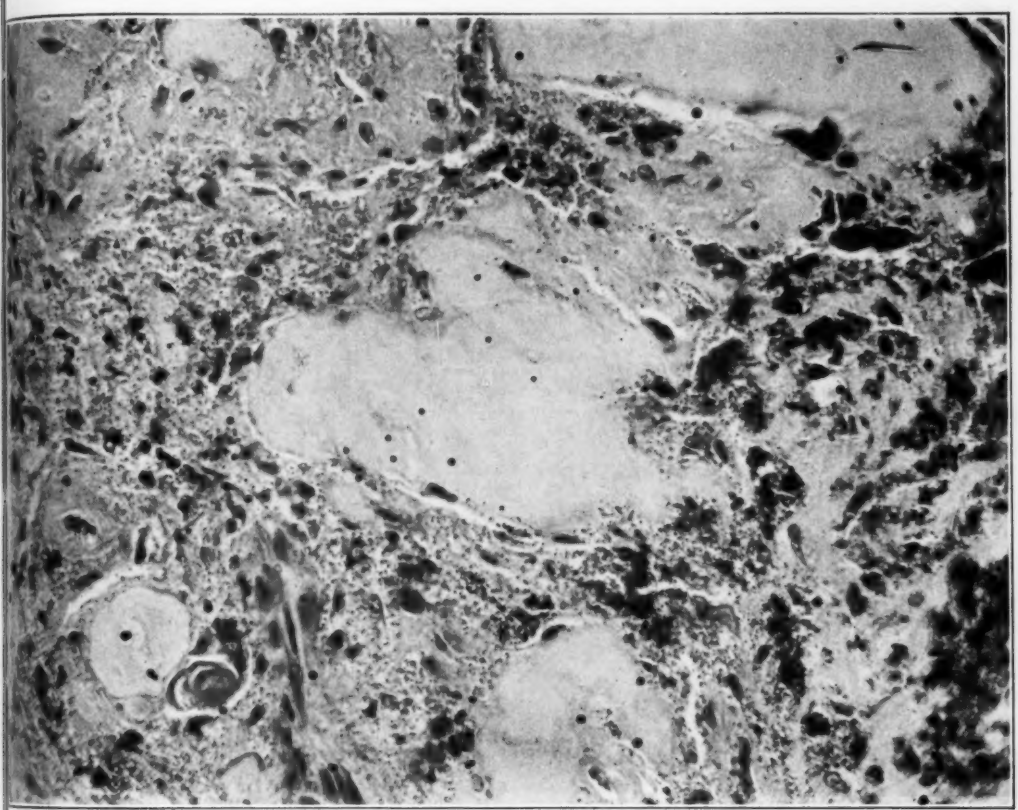

Fig. 5.—High power detail of figure 4, showing pigment-containing cells and adjacent hyaline masses.

fourth ventricle appeared normal. Section of the cerebellum and brain stem revealed no gross abnormalities. The remainder of the right optic nerve was present in a separate specimen and was evidently the portion of the nerve in and anterior to the optic foramen, since it was already encased in its dural sheath. It appeared enlarged and centrally was occupied by grayish, slightly translucent tissue in an oval area, which compressed it for two fifths of its extent. Farther forward it was still larger, being approximately twice to two and one-half

times its size near the foramen. More than half the zone was occupied by gray, granular tissue; only a crescentic band of nerve remained grossly intact.

Microscopic study of the tumor showed that it consisted of small cells with dark-staining nuclei (fig. 7), areas of calcification and occasional formation of typical rosettes (fig. 8), especially in the tumor tissue present in the optic nerve.

The pathologic diagnosis was neuroepithelioma, arising apparently from the right optic nerve or the optic chiasm.

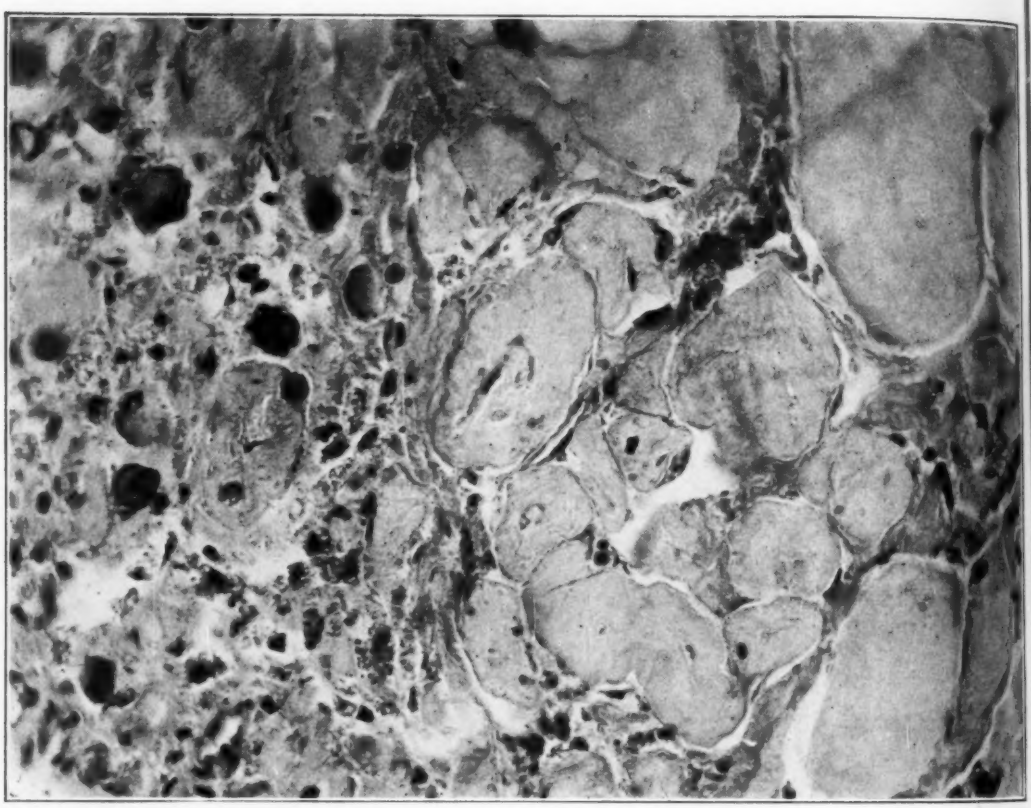

Fig. 6.—Another high power detail of figure 4, showing central endothelium-lined lumen of some of the hyaline structures and psammoma bodies.

COMMENT

An interpretation of this extraordinary case is difficult. The orbital melanosis of the optic nerve was distinct from the neuroepithelioma and arose in the sheat of the nerve. Perhaps the neuroepitheloma gave rise to some product which passed forward into the sheath of the right optic nerve and stimulated the production of the pigmented tumor.

The occurrence and distribution of melanotic cells in the leptomeninges have been carefully studied by Baader.[1] Pigment-containing cells are found normally in the pia covering the optic nerves. Excessive pigmentation (diffuse melanosis) of the leptomeninges of benign type and associated with multiple pigmented nevi of the skin has been described by Neubuerger, Daniels and Draper,[2] who cited 5 other

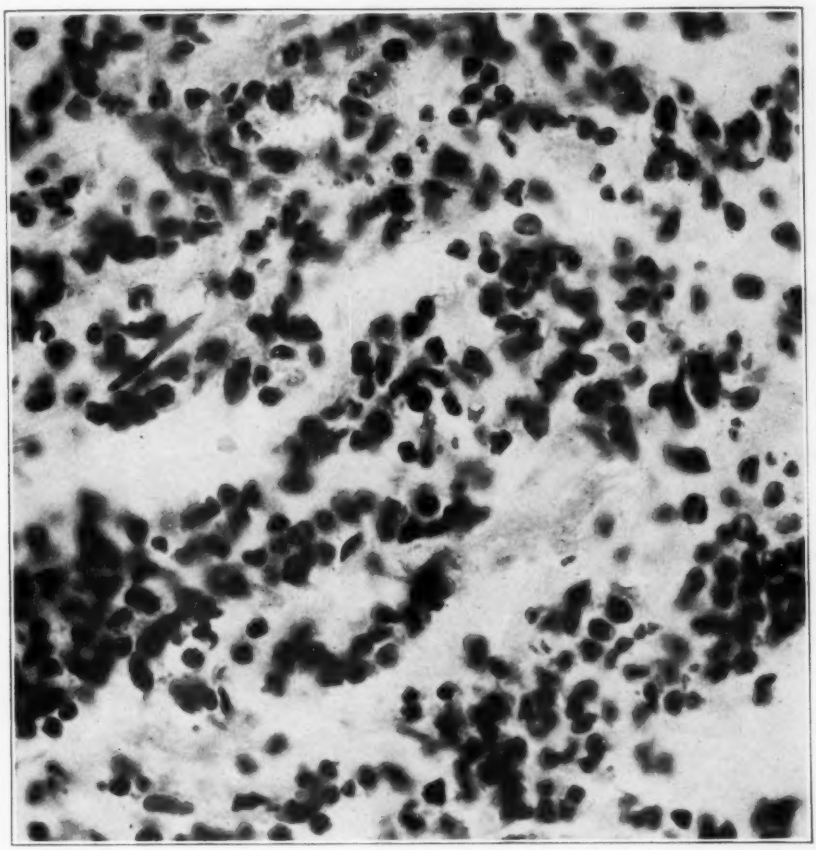

Fig. 7.—Photomicrograph showing appearance of cells of the tumor which infiltrate the base of the brain.

authors reporting cases of the same association (Rokitansky, Netherton, Grahl, Wilcox and Björnboe). To these cases may be added 2

1. Baader, O.: Ueber die Piamelanose, Ztschr. f. Zellforsch. u. mikr. Anat. **22**:735, 1935.

2. Neubuerger, K. T.; Daniels, L. E., and Draper, P. A.: Nevocytoblastoma of Brain and Meningo-Cutaneous Melanomatosis, J. Neuropath. & Exper. Neurol. **2**:140, 1943.

similar cases reported by Christensen.[3] Rottino and Kelly [4] described a case of malignant orbital melanoma and collected 43 other cases from the literature. If one can credit Theobald's [5] hypothesis that melanomas of the choroid arise from the Schwann cells of the ciliary nerves, one might propose a similar origin from the Schwann cells of the orbital nerves for the intraorbital melanomas. A more recent case has been

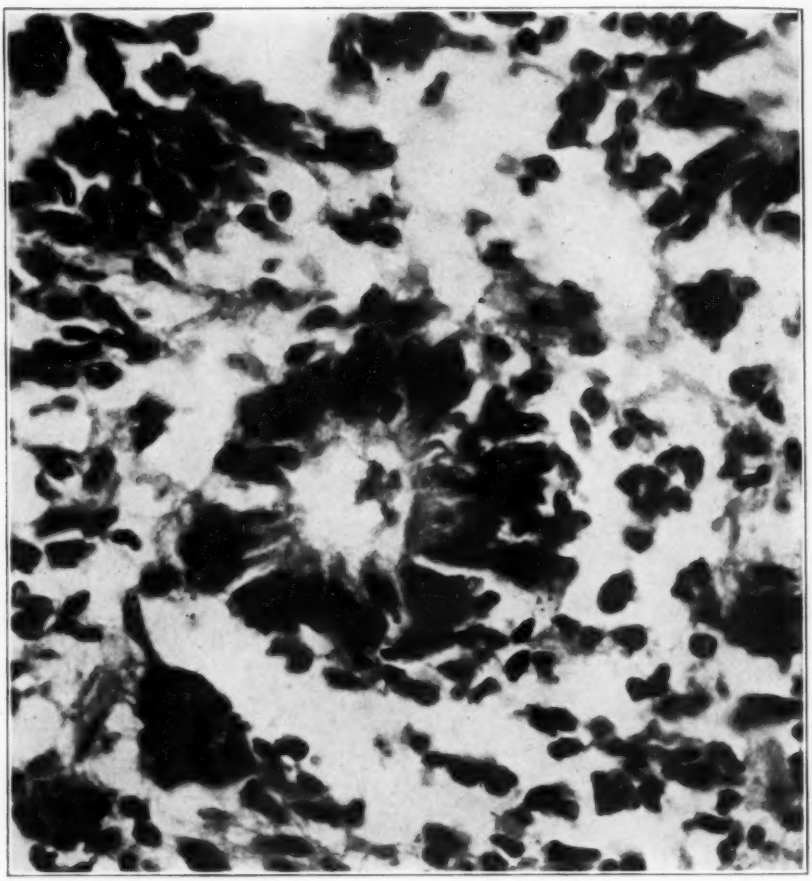

Fig. 8.—Photomicrograph of a typical rosette found in the tumor of the right optic nerve adjacent to the chiasm.

3. Christensen, E.: Primary Intracranial Melanoma: Two Cases, Acta chir. Scandinav. **85**:90, 1941.

4. Rottino, A., and Kelly, A. S.: Primary Orbital Melanoma: Case Report with Review of Literature, Arch. Ophth. **27**:934 (May) 1942.

5. Theobald, G. D.: Neurogenic Origin of Choroidal Sarcoma, Arch. Ophth. **18**:971 (Dec.) 1937.

reported by Foster.[6] A number of primary malignant melanomas of the leptomeninges have been recorded, and some of these have extended along the optic nerves (Farnell and Globus[7]; Schnitker and Ayer[8]).

Multiple malignant neoplasms of the eye and nervous system have been described. Asbury and Vail[9] reported a case of malignant melanoma of the choroid and glioblastoma multiforme of the cerebrum.

Bailey and Bucy[10] designated all the tumors arising in the meninges as meningioma with some qualifying adjective. Consequently they spoke of a melanoblastic meningioma, but this is what others call a malignant melanoma. The tumor in Cid's[11] case, arising apparently in the meninges of the restiform body, is unique, since it was made up of laminated fibrous elements resembling those of Darier's dermatofibroma and of melanoblasts. Cid explained it as a nevoid melanoma which had arisen from pial melanoblasts and formed both schwannian structures and epithelioid melanoblasts.

In view of the bizarre forms which melanotic growths of the meninges can assume, it seems to us wise to use the term "melanosis of the optic nerve" rather than "melanotic meningioma."

SUMMARY

We present the case of a 2 year old Armenian girl with glaucoma of the right eye, melanosis of the right optic nerve and neuroepithelioma of the right optic nerve, the optic chiasm and the base of the brain. Complete pathologic studies of the involved tissues are reported. The literature dealing with melanotic lesions of the leptomeninges is discussed.

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6. Foster, J.: Encapsulated Orbital Melanoma, Brit. J. Ophth. **28**:293, 1944.

7. Farnell, F. J., and Globus, J. H.: Primary Melanoblastosis of the Leptomeninges and Brain, Arch. Neurol. & Psychiat. **25**:803 (April) 1931.

8. Schnitker, M. T., and Ayer, D.: Primary Melanomas of Leptomeninges: Clinicopathological Study with Review of Literature and Report of Additional Case, J. Nerv. & Ment. Dis. **87**:45, 1938.

9. Asbury, M. K., and Vail, D.: Multiple Primary Malignant Neoplasms: Case of Malignant Melanoma of Choroid and Glioblastoma Multiforme of Right Cerebral Hemisphere, Am. J. Ophth. **26**:688, 1943.

10. Bailey, P., and Bucy, P. C.: The Origin and Nature of Meningeal Tumors, Am. J. Cancer **15**:15, 1931.

11. Cid, J. M.: Meningioma neuronevo-melanoblástico. An. de cir. **7**:137. 1941.

Clinical Notes

USE OF PENICILLIN OINTMENT IN EXTERNAL OCULAR CONDITIONS

LEO L. MAYER, M.D., ST. LOUIS

ABOUT six months ago, Eli Lilly and Company supplied me with ⅛ ounce (3.8 Gm.) tubes of penicillin ointment for trial. The ointment contained 1,000 units per gram in the form of the calcium salt and, in addition, methiolate in 1 : 10,000 concentration. A caution on the label stated, "New drug—limited by federal law to investigational use. Store below 10 degrees C. (50 degrees F.)."

This paper may be considered a preliminary report of observations on 100 patients at the time the ointment was prescribed, although for some conditions which failed to respond to the penicillin other medication was indicated.

Approximately one-half the patients were treated in the eye clinic of the Jewish Hospital. The patients and their conditions may be classified as follows:

Chronic conjunctivitis—no pus	10
Infected hordeolum	26
Infected chalazion	15
Acute nonpurulent conjunctivitis	21
Acute purulent conjunctivitis	28
	100

It should be stated that in cases of infected hordeolum and infected chalazion there was no attempt to incise or curet the abscess; instead, the penicillin ointment was used merely in external application.

The bacteriologic features of the infections were not made the subject of laboratory investigation, for two reasons: (*a*) By the time the majority of patients with conjunctivitis are seen by the physician, the infection, having been present for many days, is of mixed type with one organism seldom predominating; (*b*) infection of the glands is notoriously of the staphylococcic variety.

The results of treatment were as follows:

1. No patient with chronic conjunctivitis received any benefit from the penicillin ointment, even after two to three months of treatment.

2. In all but 1 of the patients with an infected hordeolum the infection subsided in from three to four days. Also, it was not necessary, except in the case of the 1 patient, to incise and curet the abscess.

3. In every patient with chalazion the infection subsided in from four days to one week. However, it was necessary in 13 of the 15 patients to incise and curet the gland before the lid returned to normal.

4. In no patient with the nonpurulent type of acute conjunctivitis was there the usual response and cure of the condition in a seven day period, and in all cases other treatment was necessary after the use of penicillin in order to complete a cure.

5. In all but 2 of the 28 patients with acute purulent conjunctivitis a cure was effected in from three to five days. In the remaining 2 patients it was later discovered that, owing to a nasal condition, the nasolacrimal flow was blocked, with infection of the tear ducts. When this condition was remedied, there was prompt recovery from the acute purulent conjunctivitis with further use of penicillin.

It is to be noted that superficial infections of the skin around the lids cleared dramatically with simple application of the penicillin ointment to the surface of the skin.

A further investigation is contemplated in which a study of the offending bacteria will be made.

634 North Grand Boulevard.

News and Notes

EDITED BY DR. W. L. BENEDICT

SOCIETY NEWS

Central Illinois Society of Ophthalmology and Otolaryngology.—The fourth meeting of the Central Illinois Society of Ophthalmology and Otolaryngology was held at the Hotel Orlando, Decatur, Ill., on April 27 and 28, 1946.

The following papers were presented: Saturday, April 27: "Perforating Ocular Injuries," Dr. Perry E. Duncan; "A Full Plastic Eye Implant" and "Exophthalmos," Dr. A. D. Ruedemann; "Diagnosis and Treatment of Ménière's Disease" and "Selection of Cases for the Fenestration Operation in Otosclerosis," Dr. Henry L. Williams.

Sunday, April 28: "Treatment of External Otitis," Dr. Meredith Ostrom; "Headaches from the Standpoint of the Otolaryngologist," Dr. H. L. Williams; "Treatment of Lesions of the Cornea," Dr. A. D. Ruedemann.

At the business meeting, at 1 p. m. on April 28, Dr. Stuart Broadwell Jr. was elected president for 1946; Dr. Walter Stevenson, president elect; Dr. Clarence E. McClelland, vice president; and Dr. William F. Hubble, secretary-treasurer.

GENERAL NEWS

American Orthoptic Council: Examinations for Technicians.—The next examination by the American Orthoptic Council will he held in September and October 1946.

The written examinations will be held at various cities in the country on Friday, September 6. Only those passing the written examinations will be permitted to take the oral and practical tests, to be given in Chicago, Saturday, October 12.

Applications on official forms must be received before July 1, 1946.

Address the American Orthoptic Council, 23 East Seventy-ninth Street, New York 21.

Oxford Ophthalmological Congress.—A meeting of the Oxford Ophthalmological Congress will be held at the department of human anatomy, Oxford University, July 4 to 6, 1946.

Accommodation has been secured at Keble College, and the warden has kindly agreed to include lady members as residents in college. Members will meet informally at supper on Wednesday, July 3, at 7:45 p. m., in the Hall of Keble.

The congress will open with a discussion on amblyopia, which will be led by Mr. Philip Jameson-Evans and Mrs. Dorothy Campbell.

The Doyne Memorial Lecture will be delivered by Prof. Arthur J. Ballantyne and will be entitled "The State of the Retina in Diabetes Mellitus."

Facilities will be provided in the museum for demonstrations, and members are invited to bring forward cases, specimens, instruments, apparatus and other material of ophthalmologic interest.

The council has decided that orthoptists shall be granted the privilege of attending the congress as visitors, on the invitation of a member, at sessions in which subjects affecting their department are to be discussed.

Visitors may be introduced only if sponsored and paid for by a member, after formal application to the honorary secretary and after sanction has been granted by the council.

The full program of the congress will be issued in June to all members in Great Britain and Ireland and to such members overseas as request it. Subscriptions should not be sent until the full program is received.

Basic Course in Ophthalmology, The Post-Graduate School of the University of Southern California School of Medicine.—The Post-Graduate School of the University of Southern California School of Medicine is presenting a six months' basic course in Ophthalmology, starting May 20, 1946. Dr. A. Ray Irvine, professor of ophthalmology, is director of the course, and he is assisted by Dr. Maurice Beigelman, Dr. William Endres and Dr. S. Rodman Irvine.

The entire first month is devoted to preclinical courses organized by the following men: anatomy, Dr. Paul R. Patek; physiology, Dr. Douglas Drury; pathology, Dr. Ernest Hall; bacteriology, Dr. John F. Kessel; pharmacology, Dr. Clinton Thienes; biochemistry, Dr. Harry Deuel Jr. During the second month mornings are devoted to histopathology and afternoons to intensive work in physiologic optics. The work of the third, fourth and fifth months stress clinical ophthalmology and refraction.

Clinical instructors include Dr. C. H. Albaugh, Dr. Louis Bloomberg, Dr. John Bullis, Dr. Daniel B. Esterly, Dr. Channing Hale, Dr. Deane C. Hartman, Dr. W. C. Irvine, Dr. Raphael Koff, Dr. George B. Landegger, Dr. John P. Lordan, Dr. Henry R. Nesburn, Dr. Robert A. Norene, Dr. M. W. Nugent, Dr. Stephen Popovich, Dr. Helen Preston, Dr. Alfred R. Robbins, Dr. Carrol Weeks, Dr. Clinton A. Wilson, Dr. Warren A. Wilson, Miss Dorothy Franklin, Mr. Russell Stimson and Miss Dorothy Bergin. Dr. Dwight Trowbridge, of Fresno, Calif., will be responsible for the section on neuro-ophthalmology.

The sixth month will be spent in review, with emphasis on preparation for the examinations of the American Board. Problem cases in refraction and therapeutics will be presented. Several nationally known ophthalmologists, among them Dr. Phillips Thygeson and Dr. Meyer Wiener, will be available during this final month for short series of lectures.

Examinations are to be given at the end of each month. Sixteen students are enrolled, all being returned service men. Enrolment was limited mainly by restricted facilities for work in refraction.

Cooperation of the administrative and ophthalmologic staffs of the Los Angeles County Hospital is appreciated as essential for the success of the course.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

General Diseases

OCULAR TUBERCULOSIS. D. LIVRAMENTO PRADO, Arq. brasil. de oftal. **5:** 53 (April) 1942.

Before presenting an interesting clinical observation on tuberculosis of the choroid and retina, the author summarizes the symptoms of this disease in the various parts of the eye, commenting on the many and varied treatments which produce results in specific cases. Treatment with tuberculin; gold, copper and chromium salts, and light, radium and roentgen rays is discussed. A detailed clinical observation is given; eight figures in black and white and two colored plates accompany the article.

M. E. ALVARO.

OPHTHALMOSCOPIC EXAMINATION IN CASES OF ARTERIAL HYPERTENSION. F. AYRES, Arq. brasil. de oftal. **7:** 95 (June) 1944.

In his lengthy article, the author stresses the importance of an ophthalmoscopic examination in cases of arterial hypertension, pointing out that such an examination is often of the greatest value to the general practitioner. The two types of hypertension according to Volhard, (*a*) essential hypertension, red and solitary, and (*b*) pale, or malignant, hypertension, are discussed. The ophthalmoscopic pictures encountered, including discoloration of the arteries, central reflex from the vessels, changes in arterial caliber, alterations in the course of the vessels, the double white contour, exaggerated arterial pulsation and arteriovenous crossings, are described. Besides the vascular signs, retinal lesions, retinal hemorrhages, edema, white streaks and vascular retinopathies are dealt with in detail. Four figures and bibliography accompany the article.

M. E. ALVARO.

Glaucoma

CONCEPT OF PREGLAUCOMA AND ITS DIAGNOSIS. A. MOREU, Arch. Soc. oftal. hispano-am. **4:** 313 (May-June) 1944.

The author designates as a preglaucomatous eye one which is normal but on special examination exhibits some peculiarities. He makes this examination on all patients over 50 who consult him for vague visual disturbances, and he has been successful in averting the onset of glaucoma in 25 per cent of them.

The examination includes the following procedures: (1) biomicroscopic observation for changes in the small ciliary vessels near the limbus; (2) gonioscopic examination for pigmentary changes at the angle; (3) study of pupillary reactions; (4) observations on the

fundus; (5) determination of venous permeability of the eye; (6) study of the relationship of the retinal and the systemic blood pressure; (7) determination of the light threshold and studies of the light sense with the use of the adaptometer and campimeter.

A detailed description of the procedure in all these examinations is given. The article is illustrated.

H. F. CARRASQUILLO.

Injuries

SEQUESTROMY IN GUNSHOT OSTEOMYELITIS OF THE ORBIT. B. RADZIKHOWSKY, Vestnik oftal. **23:** 34, 1944.

In the material of the N. ophthalmic evacuation hospital, osteomyelitis was observed in 20 per cent of cases of gunshot injuries of the bones of the orbit. Sequestromy was performed in 31 cases of injuries of the orbit; in 20 of these cases the lateral wall, in 4 cases the upper wall and in 7 cases the lower wall of the orbit was injured.

Radzikhowsky believed that sequestromy is the method of choice for treatment of osteomyelitis due to gunshot injury. The operation should be performed after the acute inflammatory symptoms disappear, i. e., not earlier than six weeks after the injury. Both the sequesters and the necrotic parts of the bone should be removed. Any fistula which does not heal after a prolonged period should be carefully examined for sequesters. The rhinologist's assistance is valuable in cases of injury of the lower or the upper wall of the orbit.

In cases in which injury of the cranial bones is suspected the operation should be performed by a neurosurgeon.

O. SITCHEVSKA.

Lacrimal Apparatus

DACRYOCYSTITIS OF PEACETIME AND WARTIME. V. P. STRAKHOV, Vestnik oftal. **23:** 4, 1944.

Strakhov believes that both dacryocystorhinostomy and extirpation of the sac should retain their place. He gives a number of contraindications to dacryocystorhinostomy, in cases of which extirpation of the sac is indicated instead. Epiphora in wartime is frequently due to injury of the lids at the inner angle, and the greatest care should be taken in uniting perfectly the margins of the canaliculi.

During 1942 and 1943 101 operations for dacryocystitis were performed in N. evacuation hospital; in 70 per cent of these dacryocystorhinostomy and in 30 per cent extirpation of the sac was done. In some of the cases of traumatic origin it was difficult to find the sac because of the presence of thick scar tissue; in cases of deformity of the bone it was necessary to enlarge the bony passage into the nose. Extirpation of the sac was done in cases of diseased sinuses, of syphilis (2), of trachoma (1) and of fistula and phlegmon of the sac (2).

O. SITCHEVSKA.

Lens

TRAUMATIC LENTICONUS POSTERIOR. E. ROSEN, Brit. J. Ophth. **29:** 370 (July) 1945.

Rosen gives the data in 2 cases and recalls 1 other case of traumatic posterior lenticonus.

A man aged 23 received a penetrating injury to the left eye after an explosion. A nonmagnetic foreign body entered at the corneoscleral margin, leaving a calcium-like scar. The portion of the iris corresponding to the injured sector was atrophic, detached from its peripheral union and tremulous. There was adhesion of the iris to the anterior capsule in the same meridian. The path of the projectile could be followed through the thickness of the lens to the posterior capsule, where there was a dense, almost chalky white, deposit with a metallic luster. The entire posterior cortex seemed encrusted with small white, round, saltlike deposits. The posterior capsule seemed to bulge posteriorly. Similar formations were seen in the vitreous.

The second case was one of chalcosis lentis associated with posterior lenticonus. The posterior capsule bulged posteriorly, and the posterior capsule appeared to be "rolled up" on itself in two regions, much like glass leisen bodies in the anterior chamber.

W. ZENTMAYER.

Lids

REPORT OF FORTY-EIGHT CASES OF MARGINAL BLEPHARITIS TREATED WITH PENICILLIN. M. E. FLOREY, A. M. MCFARLAN and I. MANN, Brit. J. Ophth. **29:** 333 (July) 1945.

Forty-eight patients with blepharitis were treated with local application of penicillin ointment containing 600 to 800 units of penicillin per gram of base. Staphylococcus aureus was isolated from the lesions of 39 of 41 patients examined bacteriologically. Thirty-six of the patients applied their ointment regularly three or four times a day for as long as it was considered necessary (i. e., three to ten weeks). Recovery took place in all these patients without removal of other foci of infection or any adjuvant treatment other than epilation of a few lashes. Bacteriologic observations were found to be closely associated with clinical signs, but the disappearance of Staph. aureus from cultures of material from the lids was considered a better indication for cessation of treatment than clinical signs as it invariably succeeded clinical improvement. A follow-up study a year after treatment was discontinued revealed that two thirds of the patients reporting had remained free of recurrence without further treatment.

W. ZENTMAYER.

TWO RARE CASES OF HOMOPLASTIC SURGERY OF THE EYELIDS. N. I. SHIMKIN, Brit. J. Ophth. **29:** 363 (July) 1945.

Shimkin reports 2 cases in which homoplastic surgical treatment of the eyelids was successful.

In the first case post-trachomatous trichiasis in a hemophilic youth was treated with a graft from the buccal mucous membrane of his father. The second case was one of ichthyosis ectropion in a baby of 13 months suffering from congenital generalized ichthyosis. All four lids were affected. The ectropion was successfully treated with whole skin grafts from the forearm of his mother. No case similar to the first one was found in the literature, but Elschnig reported a case similar to the second.

The article is illustrated.

W. ZENTMAYER.

Ocular Muscles

HETEROPHORIA AND NEUROSIS IN FLYING PERSONNEL. H. C. BECCLE and E. H. KITCHING, Brit. J. Ophth. **29:** 125 (March) 1945.

The functional nature of heterophoria, the variable presence of symptoms, the inconstant relationship of symptoms and signs and the presence of associated nervous symptoms and indications of emotional instability, all suggest the existence of important psychologic factors in heterophoria, calling for investigation. With this end in view, 57 successive cases of hyperphoria in flying personnel were studied; 50 of the men were suffering from a well defined psychologic illness—either anxiety hysteria or hysteria. The conclusion, however, was that the heterophoria, though often the most prominent presenting symptom, was only one manifestation of a generalized psychologic illness, which was itself the main and primary disability. The results suggest that a psychologic examination is an essential part of the investigation of ocular imbalance. Local treatment should not be started until the presence and degree of the psychoneurosis have been determined.

W. ZENTMAYER.

MONOCULAR PARALYSIS OF BOTH ELEVATOR MUSCLES. J. MALBRÁN, Arch. de oftal. de Buenos Aires **19:** 392 (Oct.) 1944.

Unilateral paralysis of both elevator muscles of the eye is not exceptional. The author has seen several cases in a comparatively short period. Its detection, however, is not common among ophthalmologists, since the methods of diagnosis are not sufficiently emphasized.

The lesion producing such a paralysis may be peripheral, in the nerve fibers of the oculomotor nerve; central in the neighboring nuclei, or supranuclear, in the motor fibers before they reach the nuclei in the mesencephalon. The first two locations are chiefly of theoretic importance, the lesion being almost always supranuclear.

The author gives a detailed description of the function of the elevator muscles and stresses the point that knowledge of their action in the various directions of gaze will lead to diagnosis of their paralysis. When a supranuclear paralysis occurs, Bell's phenomenon persists. This sign, of great diagnostic value, is, however, not mentioned by most authorities.

The surgical treatment of the condition is given. The article is illustrated with diagnostic photographs from 7 cases.

H. F. CARRASQUILLO.

Parasites

HYDATID CYST OF THE ORBIT DIAGNOSED AS SARCOMA. M. ARNAU MAORAD, Arch. Soc. oftal. hispano-am. 4: 387 (May-June) 1944.

The author was called by an otorhinolaryngologist to assist in an exenteration of the right orbit of a girl 18 years of age supposedly suffering from a sarcoma of the orbit, which had also invaded the maxillary sinus. The condition had begun one and a half years before with dull, continuous pain deep in the orbit, slight protrusion of the globe and diplopia. Three months later the pain was worse, vision began to fail and the proptosis increased. At the time of operation the pain was intense, the exophthalmos was extreme and the cornea was

necrosed. There was also a large tumefaction over the maxillary sinus. A diagnosis of sarcomatous tumor of the orbit was made.

Exenteration of the right orbit was undertaken. When the needle was inserted in the upper and inner angle of the orbit to infiltrate the tissues with the anesthetic, about 60 cc. of a clear crystalline fluid was evacuated. The diagnosis of hydatid cyst was made, and simple enucleation of the globe was performed.

The author recommends that whenever the etiologic diagnosis of unilateral exophthalmos has not been clearly established, an exploratory puncture should be made before an operation is undertaken. Photographs of the patient appear in the article. H. F. CARRASQUILLO.

The Pupil

ADIE'S SYNDROME. C. GARBINO and C. CASTELES, Arch. de oftal. de Buenos Aires **19:** 199 (April-May) 1944.

The authors report a case of Adie's syndrome (encephalomyelopathy of unknown origin), the second to be recorded in Uruguay. The patient was a nervous woman aged 35. She suffered from asthma, and in the routine examination the syndrome was incidentally discovered. She showed tonic pupils and generalized loss of tendon reflexes. The bilaterality of the ocular symptom and the generalized loss of tendon reflexes were peculiarities of the case. The serologic reactions for syphilis were negative. After an exhaustive study of the literature, the authors discuss fully the etiopathology and the pathologic physiology of the syndrome. An extensive bibliography accompanies the article.

H. F. CARRASQUILLO.

Retina and Optic Nerve

TIME FOR OPERATION FOR RETINAL DETACHMENT. H. ARRUGA, Arch. Soc. oftal. hispano-am. **4:** 205 (March-April) 1944.

With the use of diathermy in the treatment of retinal detachment it is not always necessary to operate immediately as was formerly taught by Gonin. When there is pronounced bullous separation of the retina, diathermy is often unsuccessful. Preoperative treatment should be given to allow the retina to be replaced. In summary, Arruga states: Operate at once in cases in which the retina is close to the choroid at the level of the tears, in cases of disinsertion and in cases in which the condition remains stationary or is progressive and the macula, though not yet involved, is menaced. Postpone the operation in cases in which there is bullous separation at the site of the tears or in which the detachment is localized or increased when the anatomopathologic conditions do not justify it. H. F. CARRASQUILLO.

Tumors

DIAGNOSIS OF OCULAR TUMORS BY MEANS OF BOTELHO'S REACTION TO AQUEOUS HUMOR. H. POVOA and A. PAULO JR., Rev. brasil. de oftal. **2:** 193 (June) 1944.

Povoa and Paulo Jr. performed the Botelho test for cancer on several persons in three small groups: (1) patients with nonmalignant

disease of the eye (detachment of the retina, acute glaucoma, chorioretinitis and sclerochoroiditis), (2) normal persons and (3) patients with malignant tumors of the eye. The diagnosis of malignant tumor was made from the results of biopsy in 1 case and without a biopsy in 3 cases. The Botelho test gave negative results in the aqueous humor of the patients with nonmalignant disease of the eye, as well as in the aqueous of normal persons. It gave positive results in all 4 cases of malignant ocular tumor. The results of histopathologic examination of the eyes after enucleation confirmed those of the Botelho test. This paper is a preliminary report. The authors point out the advisability of performing the test in all cases of tumors posterior to the crystalline lens which are not amenable to biopsy and which are suspected of being malignant.

W. ZENTMAYER.

Uvea

GLAUCOMA AND ESSENTIAL PROGRESSIVE ATROPHY OF THE IRIS. H. S. SUGAR, Am. J. Ophth. **28:** 744 (July) 1945.

A case of progressive atrophy of the iris with increased intraocular pressure is reported. Endothelial involvement and increased density of the iris at the area toward which the pupil was drawn were observed. The presence of increased intraocular pressure was explained by the dense peripheral anterior synechias. No attempt is made at present to explain the cause of the atrophy of the iris.

W. S. REESE.

Vision

INFLUENCE OF SELECTED SPECTRAL DISTRIBUTION ON THE GLARE EFFECT STUDIED BY MEANS OF DARK ADAPTATION. E. SIMONSON, S. BLANKSTEIN AND E. J. CAREY, Am. J. Ophth. **28:** 712 (July) 1945.

In 3 trained subjects the dark adaptation of the cones proceeded faster after exposure to an illuminant (B) whose spectral range had a reduction at each end of the visible spectrum than after exposure to the usual frosted lamps (illuminant A). The difference was not significantly influenced by four levels of preadaptation brightness, by daily variations or by individual variations. Evidence is presented that only the speed of dark adaptation is changed, without essential change in the contour of the curves. In a significant majority of 32 untrained subjects, the dark adaptation of the cones was faster after exposure to lamp B, except the first readings at 10 millifoot candles where no significant difference after exposure to lamp A and exposure to lamp B was observed. The difference in dark adaptation for lamp A and lamp B was about the same in four groups of 8 subjects each, grouped according to the individual speed of dark adaptation. The usefulness of dark adaptation experiments for the appraisal of the glare effect is discussed.

W. S. REESE.

Therapeutics

PENICILLIN THERAPY IN OCULAR INFECTIONS. J. G. BELLOWS, Am. J. Ophth. **27:** 1206 (Nov.) 1944.

Bellows gives a summary and draws the following conclusions:

"1. Penicillin reaches the ocular tissues within a few minutes after intravenous injection.

"2. After a large dose of penicillin is administered intravenously, it appears in the ocular tissues listed in decreasing order of concentration as follows: extraocular muscles, sclera, conjunctiva, blood, tears, chorioretinal layer, aqueous and vitreous humors, and cornea. It has never been detected in the crystalline lens. In this respect, the lens is similar to the cerebrospinal fluid, brain, and nerve tissues.

"3. Local application of penicillin leads to a very high concentration of the drug in the tissues of the anterior segment of the globe.

"4. The following four ointments, in which the penetrability of penicillin was tested, are listed in the order in which they are clinically recommended: simple ointment, oil-in-water emulsion, and lubricating jelly. The 'vanishing' stearate type of a base, in which penicillin seems to have the greatest power of corneal penetration, is not recommended because of its possible damage to the corneal epithelium. However, it may be used on the skin of the lids.

"5. Penicillin was found to be effective in the clinical treatment of acute and chronic infections of the lids, conjunctiva, and cornea produced by penicillin-sensitive organisms.

"6. It was found ineffective in two cases of exudative choroiditis of undetermined origin and in one case of gonorrheal iridocyclitis.

"7. Susceptible individuals may become hypersensitive to penicillin."

W. S. REESE.

PENICILLIN IN THE TREATMENT OF PERFORATING OCULAR INJURIES AND IN UVEITIS, R. G. SCOBEE, Am. J. Ophth. **28:** 380 (April) 1945.

Scobee concludes from experiments that only instillation of penicillin together with intravenous injection of the drug is at all satisfactory in controlling severe ocular infection. He tried lavage of the anterior chamber, subconjunctival injection, use of eye drops and intravenous injection. He believed that penicillin is a definite adjunct to the therapy of uveitis.

W. S. REESE.

PENETRATION OF PENICILLIN INTO THE EYE. R. E. WRIGHT, and C. H. STUART-HARRIS, Brit. J. Ophth. **29:** 428 (Aug.) 1945.

The authors emphasize that their observations are incomplete because work was interrupted by circumstances beyond their control. It is felt, however, that observations on the human aqueous humor were of sufficient interest to record, particularly as they tended to support the results of von Sallmann and Meyer obtained with animals.

The following summary is supplied:

Observations designed to indicate the most effective method of introducing penicillin into the eye are recorded. Penicillin reached the aqueous humor after local use in the conjunctival sac or after intramuscular injection, but the concentrations attained are variable and often low. By the use of iontophoresis, higher intraocular concentration can be obtained, but the technic requires considerable care and attention.

W. ZENTMAYER.

PENICILLIN IN OPHTHALMOLOGY. T. G. W. PARRY, G. C. LASZLO and J. L. PENISTON, Brit. J. Ophth. **29:** 479 (Sept.) 1945.

This study has been largely directed toward ascertaining the value of penicillin in self administration by patients in their own homes. Methods of application, the use of penicillin for more severe conditions and some points of laboratory technic are also recorded.

The application of cream seems to be so far the only method of outpatient treatment. A preparation of standard strength of 250 units per gram dispensed in a screw-capped applicator tube in small quantity is employed.

W. ZENTMAYER.

INDICATIONS FOR RETROBULBAR INJECTION OF ALCOHOL. A. PAULO FILHO and J. DE ARRUDA, Rev. brasil. de oftal. **2:** 37 (March) 1944.

Paulo Filho and de Arruda have for several years successfully resorted to the retrobulbar injection of alcohol in the therapy of acute ocular pain accompanying absolute glaucoma and acute hypertensive uveitis, as well as for the control of acute ocular and orbital pain in cases of ophthalmic herpes zoster, interstitial keratitis and acute ocular hypertension due to post-traumatic hemorrhage into the anterior chamber. The treatment consists of a first injection of 1 cc. of a 1 or 2 per cent solution of procaine hydrochloride and, two or three minutes later, and without removal of the injecting needle, a second injection of 1 cc. of alcohol, either absolute or of 80 per cent strength.

W. ZENTMAYER.

Society Transactions

EDITED BY DR. W. L. BENEDICT

COLLEGE OF PHYSICIANS OF PHILADELPHIA
SECTION ON OPHTHALMOLOGY

Warren S. Reese, M.D., Chairman

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Oct. 18, 1945

Neurofibromatosis Occurring in Three Consecutive Generations: Report of a Case. DR. EDITH HARVEY (by invitation).

A girl 5 years of age was seen in the clinic, with a history of gradual protrusion and deviation downward of her left eyeball. She was a healthy, intelligent child. She had had none of the usual diseases of childhood except an occasional cold. A tonsillectomy had been performed the previous year.

Examination revealed exophthalmos of 3 mm., with the visual axis directed downward 30 degrees and temporally 5 degrees when the visual axis of the normal eye was directed straight forward. Ocular rotations were fairly good in all fields except that of the superior rectus muscle, in which there was definite limitation. Visual acuity was 20/30 in the normal eye and 1/200 in the exophthalmic eye, and there was a definite papilledema in the latter. She had café au lait spots over her chest and abdomen but no subcutaneous neurofibromas.

The blood count and the urine were normal, as were the serum calcium and phosphorus.

Roentgenographic studies of the orbit showed no involvement of the bone except slight enlargement of the optic foramen on the side of the defect. The sphenoidal ridges were normal, and the bones of the skull showed no abnormalities. There was no evidence of calcification. Roentgenographic studies of the sinuses showed a normal condition.

The patient was operated on by Dr. E. B. Spaeth. A neurofibroma was found encircling the entire intraorbital portion of the optic nerve, from its exit from the eyeball to the optic foramen. The eyeball and the optic nerve up to the foramen were removed. The tumor, which was about 9 mm. in diameter along the entire length of the nerve, seemed to end at the optic foramen, where the optic nerve was of normal size; and it is to be hoped that the tumor did not involve the intracranial portion of the nerve.

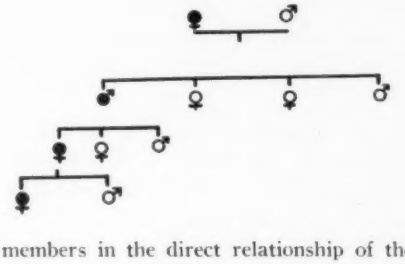
No other tumor masses were palpable in the orbit.

Microscopic sections of the enucleated eyeball and the nerve showed an intact, undamaged optic nerve, with a pure fibroma, containing no nerve fibers, surrounding it. There was, in addition, edematous infiltration of the nerve heads. (These sections will be discussed later in greater detail by Dr. Perce DeLong, under whose direction they were made and carefully studied.)

The mother of the child has multiple cutaneous neurofibromas, most numerous over her back but scattered over her abdomen, neck and extremities. Many have been present since birth; others appeared at puberty. She has a few café au lait spots on her abdomen. Microscopic diagnosis of one of the tumors on her arm was neurofibroma. A similar tumor had been removed several years previously, and the same microscopic report was made.

The child's grandfather on the maternal side had died in Presbyterian Hospital. He had had a suboccipital craniotomy for neurofibromatosis involving the middle and posterior fossae. The diagnosis at autopsy was generalized neurofibromatosis, subserous neurofibroma of the stomach and a cervical perineural fibrosarcoma.

The antecedent relatives lived in Norway, and the only history obtainable was that the child's mother recalled hearing that her own grandmother, on the paternal side, had had several nodular masses on her face. Whether these were neurofibromas is of course impossible to determine.



Affected members in the direct relationship of the patient.

The brother of the child and the sisters and brothers of the mother have no known abnormalities suggestive of neurofibromatosis.

Neurofibromatosis is a hereditary, and sometimes a familial, condition characterized by lesions of the skin and tumors on nerves. These lesions were discussed in detail. The author presented a brief résumé of the general symptoms.

The disease is congenital, showing mendelian dominance in successive generations, although occasionally a generation is skipped. There is no sign of sex-linked inheritance.

There seems to exist a congenital defect of the nerve sheath tissue, resulting in hyperplasia of the connective tissue of the nerve sheath, the cells being adult in type and not embryonic. The stimulus to hyperplasia is present from birth. New tumors may appear at puberty, during pregnancy or with intercurrent infections. They are not stimulated by local trauma.

The origin of these tumors has been a matter of controversy, which still is not settled. Von Recklinghausen expressed the belief that the tumors were mesoblastic in origin, arising from the endoneurium. Verocay stated that they were ectodermal, derived from the cells of the sheath of Schwann. According to Penfield and Young, they are the result of a reaction of connective tissue about the fibers of the nerves and show both nerve fibers and connective tissue elements, although the differentiated fibroblast, and not the glial cell, is the type cell.

Sarcomatous changes have frequently occurred in cases of neurofibromatosis. Operative removal of a neurofibroma may be followed by the recurrence of a benign neurofibroma. On the other hand, sarcomatous transformation may occur at the site of removal or in a distant preexisting nodule. Metastasis is relatively infrequent when a benign neurofibroma becomes sarcomatous; instead, such transformation is followed by transformation into sarcoma of a neurofibroma in another part of the body.

DISCUSSION

DR. PERCE DELONG: This syndrome is a disease of congenital origin characterized by cutaneous pigmentation and multiple tumors of cranial and peripheral nerves. The growths in Recklinghausen's disease are not true neuromas, which must consist of both nerve tissue and nerve cells; hence the term neurofibromatosis.

Penfield and Young, in describing the involvement of the nerve trunks, meninges and central nervous system, stated that there is a definite hyperplastic reaction of the cells peculiar to these tissues, indicating that an irritant or stimulating influence must have been exerted, causing hyperplasia, which was followed by true neoplastic growth of these cells.

This specimen does not differ from those described by other authors. The clinical ocular signs are secondary. These are exophthalmos, the result of pressure from behind; choked disk, due to pressure; degeneration, and atrophy of the optic nerve. Only van der Hoeve has reported involvement of the retina.

Pathologic examination in this case showed that the tunics of the globe were normal except for some edema of one ciliary nerve, with thickening of its sheaths. Then, too, there was early papilledema of the nerve head.

The optic nerve presents pronounced edema and definite fibrosis of the sheaths. Hyperplasia is most conspicuous in the region of the arachnoid, where the nerve has the appearance of being about twice its size. Microscopically no cellular elements are present, but pronounced hyperplasia and fibrosis of the nerve sheaths exist.

DR. E. B. SPAETH: I wish to call attention again to the development of sarcoma in cases of neurofibromatosis. I recently have seen 2 patients. The first was presented before this section about two years ago with neurofibromatosis in the upper division of the fifth nerve, and now sarcomatous changes accompanied with severe hemorrhages are present. The second patient had a neurogenic sarcoma that seemed to arise in an otherwise benign neurofibroma of the orbit.

The astonishing feature of the cases presented by Dr. Harvey is the great dissimilarity seen in 3 cases of a common hereditary condition. In the first case, that of the grandfather, there was a neurogenic sarcoma, i. e., sarcomatous changes associated with neurofibromatosis. In the second case, that of the mother, the lesions were confined entirely to the skin, and in the third case, that of the child, there was a neurofibroma of the optic nerve itself.

DR. FRANCIS HEED ADLER: I should like to ask Dr. Harvey whether this tumor did not originate in the sheath of the nerve, rather than in the nerve fibers. I should like to point out the error in the theory that neurofibromas start in the neurolemmal sheath, at least in the case of the optic nerve, because the optic nerve fibers, like the fiber tracts of the brain, have no neurolemmal sheath. The optic nerve is not a true sensory nerve.

DR. EDITH HARVEY: It is now generally believed that neurofibromas arise from the connective tissue sheaths of the nerves and not from the neurilemma; that is, they are mesodermal in origin.

Early Ocular Pemphigus: Report of a Possible Case. DR. JOSEPH V. KLAUDER (by invitation) and DR. ALFRED COWAN.

A white woman aged 29 since February 1944 had had recurring erosive areas on the buccal mucosa, the lips, under the tongue and on the floor of the mouth. These areas disappeared and reappeared at intervals of from a few weeks to a few months. When lesions appeared on the lips, she observed that the initial manifestation was a blister, which soon ruptured, leaving a crusted surface.

In July 1945, when she was first seen, she had a cutaneous outbreak. This was the first time such lesions had appeared. On each elbow there was a bleb, which was partially collapsed. The skin around the bleb was normal. There was a similar lesion near the external canthus of the right eye. The lower lip was covered with a crust; when the crust was removed a moist, bleeding surface was seen. On the mucosa of each cheek there were marginated, superficial ulcerations, the surfaces of which were covered with a yellowish membrane. There were no vesicles on the lips or in the mouth.

In August, during the time lesions were in the mouth and on the skin, both eyes became inflamed. Examination showed intense infection of the palpebral and bulbar conjunctivas. There was a distinct membrane on the tarsal conjunctiva of the lower lids and on the bulbar conjunctiva on the temporal side. Both upper lids were similarly affected. Biomicroscopic examination showed that both corneas were clear, there were no vesicles, the aqueous was clear, the iris intact and the lens unaffected.

Infection of the conjunctiva gradually subsided, and the membrane on the conjunctiva of both eyes disappeared. In the latter part of September there was a recurrence of conjunctival redness, with formation of a membrane on the upper and lower lids. There was moderate swelling of the upper lid with free discharge. No vesicles were seen in either cornea.

The patient has had no fever and no constitutional symptoms. Sulfadiazine was given internally, and sulfathiazole ointment was used in the eyes. Solution of potassium arsenite U. S. P. was administered by mouth in doses varying from 3 to 6 drops three times a day. Repeated vaccinations for smallpox were performed. A strict saltless diet was instituted.

Ocular inflammation again subsided. At the time of this report the patient presents only erosive areas on the inner surfaces of the lips and on the buccal mucosa.

DISCUSSION

DR. GEORGE J. DUBLIN: It has been my good fortune to be able to observe the course of the disease in this case from its incipience. When the patient was first seen, there was noted a severe inflammation of the conjunctiva. An extensive membrane was present on the tarsal conjunctiva of the lids. This membrane was yellowish, appeared to lie on the surface of the conjunctival epithelium and did not seem to involve the deeper layers. I tried to remove part of the membrane but was unsuccessful. Two or three days later a membrane was noted on the bulbar conjunctiva, both temporally and nasally, paralleling the limbal border, and in one eye it extended to the outer canthus. In addition, there was a profuse ropy discharge.

Dr. Klauder's observation of a previous vesicular formation in the skin, together with stomatitis, pointed to possible pemphigus. Mild lotions were prescribed, and the condition cleared to a remarkable degree in seven to ten days, at which time the membrane had completely disappeared in both eyes.

The patient volunteered the information that as soon as the eyes would become well various sores would break out in the mouth. Her statement was correct. This condition in and about the mouth lasted four or five days, and then there was a recurrence of the ocular symptoms except that this time the membrane was even more extensive than in the previous flare-up. I have never seen a case in which there was such an extensive membrane formation. I did not know how to treat the condition, as there is no adequate treatment.

Other conditions simulating pemphigus are erythema multiforme, dermatitis herpetiformis and bullous syphilids. There are still other conditions which produce membranes, of the pseudo and the deep type of membranous conjunctivitis.

Membranous conjunctivitis involves the deep portion of the epithelium or the submucous portion and is considered invariably to be due to diphtheria. This patient was not acutely ill, nor was there any rise of temperature. As no diphtherial bacilli were found, the possibility of this disease was eliminated. Material for culture and smear was taken, but the bacteriologist was unable to identify the organism, stating that it was a rare gram-positive bacillus.

The patient had two other exacerbations associated with new lesions in the mouth. No special treatment was ordered, because it was thought that the condition would clear up under simple, bland medication. The patient, however, received sulfanilamide internally in doses of 60 grains daily (3.9 Gm.) for approximately ten days. I do not feel that the disappearance of the membranes was due to the sulfanilamide therapy, because they had cleared up without it in previous attacks. I saw the patient about four days ago, when all the lesions and membranes had disappeared. It is significant that the cornea was at no time involved. There were no vesicles and no ulcers. In spite of the dense membrane which was present, the conjunctiva, both tarsal and bulbar, at no time showed any bleeding, ulcerated areas or areas of adhesion. Vision at all times was 20/20 in each eye.

Pemphigus is a rare disease. It may assume an acute or a chronic form. The acute form, occurring chiefly in butchers, is a virulent

disease, with severe constitutional symptoms, and frequently causes death. Chronic ocular pemphigus is frequently called essential shrinkage of the conjunctiva. The cause is unknown, but the ocular condition is associated with bullae in the skin and mucous membranes in various parts of the body. In the eye, vesicles are rarely seen in the conjunctiva; in their place, small reddened areas are noted, probably the end result of broken bullae. This condition is frequently incorrectly diagnosed, or at least a proper diagnosis is not made until the late stages, when shrinkage of the conjunctiva takes place. This shrinkage is due to formation of connective tissue in the submucous portions of the conjunctiva; when this occurs, there is thickening of the conjunctiva with symblepharon and, eventually, ankyloblepharon.

Biomicroscopic examination in the late stages usually reveals perivascular thickening, together with a parchment-like appearance of the conjunctiva. Vesicles in the cornea are rarely seen, but corneal changes do occur secondary to the pathologic changes in the conjunctiva. The diagnosis is made generally by a dermatologist on the finding of bullae and vesicles in the skin and in the mucous membranes of the body, especially the mouth, larynx, nose and rectum. A pseudomembrane with a chronic type of conjunctivitis is sometimes seen. The membrane affects both the tarsal and the bulbar conjunctiva, in contrast to true pseudomembrane, which affects only the tarsal conjunctiva.

In the case presented by Dr. Klauder and Dr. Cowan there were noted vesicles in the skin and in the mucous membrane of the mouth, with a history of numerous exacerbations. The ocular changes consisted of membranous conjunctivitis, with numerous flare-ups and with complete disappearance of this membrane in seven to ten days. Although the conjunctiva has not shown any permanent defect, in all probability there will be alterations. Time alone will confirm or disprove the diagnosis of pemphigus in this case.

Dr. William Lamotte, Wilmington, Del.: Dr. Klauder stated that there is no laboratory test for pemphigus. There is reported in the literature a so-called Macht test, with which I am not familiar; I wonder whether Dr. Klauder would tell us what it is and whether it is of any value?

Dr. W. E. Fry: Another differential diagnosis at times may have to be considered, namely, the difference between vernal conjunctivitis and pemphigus. I mention this because of a patient who came under observation about a year and a half ago. The patient, a yonug woman, was seen by Dr. Klauder. She gave a history of multiple lesions of the gums, which necessitated the removal of all her teeth. Exactly what the lesions were is not known.

When I saw the patient, the ocular condition was that of papillomatous formation over the palpebral conjunctiva. The symptom of which she complained was intense itching. This led me to believe that the condition was vernal conjunctivitis. The smears showed an enormous number of eosinophils. There were numerous nodules, and microscopic examination of one of them showed that the tissue was similar to that of clinical vernal conjunctivitis.

Dr. Klauder saw the patient and found evidence in the mucosa of previous lesions. He expressed the belief that the patient had pemphigus.

Since that time the patient has died. The exact cause of death I do not know except that it was not an accident; presumably Dr. Klauder's diagnosis of pemphigus is correct.

DR. WARREN S. REESE: I should like to ask Dr. Klauder whether in his experience cutaneous lesions usually occur with ocular pemphigus.

DR. JOSEPH V. KLAUDER: I do not think the Pels-Macht test is an infallible means of diagnosing pemphigus. False positive reactions are obtained, and I believe it is the consensus that the test is unreliable.

I cannot give offhand the percentage of cases of ocular pemphigus in which the disease exists without involvement of other mucous surfaces and without cutaneous lesions. It may occur without other lesions. It may accompany lesions of mucous membranes, and it may occur with lesions both of the mucous membranes and of the skin. I think, however, that in the minority of cases, I should guess in about 25 per cent, the disease is confined to the conjunctiva. In this connection, I cite Klauder and Cowan (*Am. J. Ophth.* **25:** 643-661 [June] 1942).

Herpes Zoster Ophthalmicus Appearing After Trauma. DR. JOSEPH V. KLAUDER (by invitation).

Report is made of 7 cases of herpes zoster appearing after injury to the skin or to the cornea, inflicted in various ways and of various degrees of severity. The disease appeared in the region of injury after an interval varying from one to nine days. In some of the cases symptoms of sensory disturbance or pain intervened between the injury and the outbreak of herpes zoster.

Of the 7 patients, 4 had herpes zoster ophthalmicus. The injuries preceding appearance of the disease were foreign body in the cornea, contact of the eye and forehead with sulfur dioxide and, in 2 cases, bumping one side of the head on a hard object without producing demonstrable injury to the skin.

Review of the literature disclosed many cases of herpes zoster appearing from one to thirty days after injury, at times trivial, to the skin or the cornea. Herpes zoster has also been reported as appearing after lumbar puncture, intramuscular injection of a medicament, osteopathic manipulation of the spine and high voltage roentgen radiation therapy in the region of a ganglion.

From the considerations discussed, it is reasonable that trauma may precipitate herpes zoster. The minimum degree of trauma and the interval between trauma and the appearance of herpes zoster necessary in order that trauma be ascribed a casual role cannot be precisely defined. Experimental evidence and clinical observation in cases of spontaneously occurring herpes zoster indicate that the incubation period of the disease ranges from a few days to about two weeks. In the cases of herpes zoster appearing after trauma which are reported here and in the cases recorded in the literature, an interval of one day to three weeks predominated.

DISCUSSION

DR. WALTER I. LILLIE: Dr. Klauder's presentation has interested me, inasmuch as in the cases I have reported concerning the use of smallpox vaccine in the treatment of herpes zoster ophthalmicus there has been no history of trauma. Because there has been such a tre-

mendous variation in the time of onset of the herpes after trauma in Dr. Klauder's cases, varying from less than a day to six weeks, real doubt can be raised as to the importance of the trauma. As every one knows, it is not difficult to elicit a history of trauma in almost any case of any condition. I recall a patient I saw at the Mayo Clinic with one blind eye and a very small field of vision remaining in the other due to a tumor of the pituitary gland who received $2,500 compensation when the court agreed with the patient that the visual loss was due to a pair of pliers striking his forehead. I am sure it is generally agreed that herpes zoster is a virus disease which has a definite period of incubation of a week or more and which belongs to the chickenpox family. This explains why cases of herpes zoster and of chickenpox may often be found in the same family. I should like to know what significance Dr. Klauder places on the history of trauma in his cases.

DR. GEORGE F. J. KELLY: At present I have under my care a patient with herpes zoster ophthalmicus. According to her statement, five days previous to the appearance of the lesion hot lard splashed on the involved area. My feeling is that this was coincidental, and not causative.

DR. JOSEPH V. KLAUDER: Experiments to ascertain whether herpes zoster is caused by a virus have given conflicting results. The opinion is stated by some investigators that the evidence is not conclusive and that herpes zoster may represent a syndrome resulting from any process causing inflammation of the spinal ganglion. Others accept experimental evidence as valid proof of virus causation and regard the elementary bodies demonstrable in vesicles of herpes zoster, particularly in the first forty-eight hours, as representing the specific virus of the disease. Proof of the virus causation of zoster is not so readily demonstrated as is such proof for herpes simplex.

DR. WALTER I. LILLIE: It was my opinion that Filch, in his classification of ophthalmologic conditions, placed herpes zoster in the same large family with chickenpox.

DR. JOSEPH V. KLAUDER: I am willing to do that, too.

DR. WALTER I. LILLIE: The use of smallpox vaccine in treatment of herpes zoster was not original with me, but I have forgotten the name of the one who first used repeated injections of the vaccine.

DR. JOSEPH V. KLAUDER: A virus causation of herpes zoster is the basis of this treatment.

DR. JOSEPH V. KLAUDER: What about trauma as a factor?

DR. WALTER I. LILLIE: From a review of the cases that I have had and the résumé of the literature by Edgerton I could find nothing correlating trauma with herpes zoster.

Surgical Methods of Treating Paralysis of the Superior Oblique Muscle. DR. WILLIAM E. KREWSON, III.

Isolated unilateral paralysis of the superior oblique muscle is frequently encountered; bilateral involvement, occasionally. The literature records relatively few cases of the condition treated surgically, and in these reports a great variety of procedures is offered; most of the muscles of each eye have been used in one way or another at some time. These methods include weakening of the homolateral superior

rectus, advancement of the homolateral inferior rectus, advancement or transplantation of the homolateral external rectus, weakening of the contralateral inferior rectus, tenotomy or recession of the homolateral inferior oblique, shortening of the contralateral superior rectus, advancement of the paralytic superior oblique muscle and various combinations of two or more of these operations.

No standard sequence of corrective measures can be advocated for all cases; the plan of attack must be based on measurements found in the individual case. If a choice is permitted, weakening of a depressor muscle is undesirable; strengthening of a depressor or weakening of an elevator muscle is preferable. In cases of paralysis bilateral equalization and preservation of the remaining depressor muscles are advisable.

DISCUSSION

Dr. Francis Heed Adler: I should like to discuss more fully a factor which occasionally leads to an erroneous diagnosis in cases of paralysis of the superior oblique muscle. The condition was first pointed out, I believe, by Chavasse, under the designation "inhibitional palsy of the contralateral antagonist." It is seen in cases in which fixation is customarily carried out by the eye with a paralyzed superior oblique muscle. In these cases not only is there a characteristic limitation of movement downward and to the side opposite that of the paralyzed superior oblique but there is also limitation of movement of the nonparalyzed eye on looking up to the ipsilateral side. For example, in a case of paralysis of the left superior oblique the left eye fails to move down when the eyes are turned down and to the right. In such a case the right eye fails to move up when the eyes are turned up and to the right. This condition may easily be mistaken for paralysis of the right superior rectus muscle. The explanation of this so-called inhibitional palsy, I believe, is somewhat similar to the explanation of the vertical separation on looking down and to the right when the left superior oblique is paralyzed. The latter is due to two factors: (1) the weakness in the left superior oblique muscle and (2) the overaction of the right inferior rectus muscle. This overaction is explained by the well known law of Hering, namely, that all voluntary movements are brought about by equal innervation of the muscles of the two sides concerned in the movement. When one looks down and to the right, an equal innervation is sent to all the muscles of the two sides which carry the eyes in this direction. When one of these muscles is weak, the total amount of innervation is increased so as to bring about the full movement of this weak muscle, but the same amount of innervation is sent to the yoke muscle of the opposite side, and so this muscle "overacts."

When a patient with paralysis of the left superior oblique looks up and to the right with the left eye fixing, the movement in this direction is made easier by the paralysis of the left superior oblique, because the left inferior oblique now has no resistance from its opponent. Less energy will be required, therefore, to turn the left eye up and to the right; accordingly, less innervation is sent to the yoke muscle of the left inferior oblique, namely, the right superior rectus; and this eye fails to move up as far as the left. Slides of 2 cases were presented.

Dr. Glen G. Gibson: Dr. Krewson has covered the subject completely, and the paper is a timely one. I was interested in this pre-

sentation because of the experiences which I have had in cases of mistaken palsy of the superior oblique muscle. One of the common conditions which may be incorrectly diagnosed is that which simulates convergent strabismus. In cases of such palsy the strabismus begins primarily as a paralysis of the superior oblique, but as time goes on it becomes concomitant. Such a condition is impossible to differentiate from the usual accommodative convergent strabismus. In cases of this type I have at first failed to recognize the origin of the condition in the vertical muscles and have operated as though the squint was of convergent type, only to have the patient return with an unsatisfactory result and then, on further study, to find that the paralysis was of trochlear origin. A second operation then had a gratifying result, the surgical procedure having been done in accordance with the primary pathologic condition.

Careful attention to the sensory correspondence is helpful in selecting the proper type of operation and the amount of surgical correction. This is simply illustrated by a case of traumatic trochlear palsy of one year's duration in which there was 21 prism diopters of hyperphoria. The patient was subjected to recession of the inferior rectus muscle of the unaffected eye. After the operation, which consisted of a 2 mm. recession of the inferior rectus, there was no hyperphoria. In other words, the 21 prism diopters of hyperphoria had been reduced to zero by a minimal recession. The important point in this case is not that a precise measurement of the amount of necessary surgical correction was made but, rather, that a good surgical result was achieved because this patient originally had, and always maintained, normal binocular vision until his accident. I needed only to put the eye approximately in its normal place. The sensory mechanism came to my assistance and brought the eyes to the exact place that was wanted not because the surgical correction was precisely accurate but because normal binocular vision was present before the accident.

In cases of long standing or of congenital hypertropia a recession of 2 mm. is inadequate to bring about the desired result, particularly in cases of pronounced vertical hypertropia; and in such cases it is necessary to do as much as 4 or 5 mm. of recession, and that is about the maximum amount of recession of the inferior rectus muscle of the other eye which one should do. In some cases of long standing hypertropia, particularly in adults, it is necessary to combine the recession of the inferior rectus of one eye with a tenotomy of the inferior oblique of the paralytic eye.

Another important point is the recognition of birth trauma and heredity as causative factors in trochlear paralysis. As an illustration of the hereditary factor, I recently saw two grandmothers of 2 patients on whom I had operated for trochlear paralysis. They had had the same condition all their lives. Birth trauma is a common cause, and it is fruitful to inquire particularly regarding forceps delivery as a possible cause of trochlear palsy. It is also important to consider the degree of binocular vision which the patient has. If the condition has been present from birth, the patient never had binocular vision except in abnormal positions of the head, and a much more extensive surgical procedure has to be done than in a case of traumatic origin.

Book Reviews

Eléments de gonioscopie normale, pathologique et expérimentale. By Archimede Busacca, São Paulo, Brasil: Tipografia Rosolillo, 1945. Pp. 201, with 107 illustrations (1 color plate).

Dr. Busacca is a distinguished Italian ophthalmologist and investigator who emigrated to Brazil some years ago and lately has undertaken the study of gonioscopy. This book is a valuable addition to the literature of this fast growing department of ophthalmology. Dr. Busacca has made his examinations of the angle of the anterior chamber with the Goldman contact glass, which permits observation with the patient sitting up in front of the observer, as in ordinary slit lamp microscopy. The image of the angle is seen not directly, as with the gonioscope, but indirectly, in the depth of a plane mirror. Also, the two sides of the angle appear somewhat foreshortened. The Goldman glass permits a comparatively easy observation of the upper and lower parts of the angle, but the temporal and nasal sides are difficult to illuminate and bring to focus properly. To avoid this drawback, Goldman has devised a special reduction prism, which is attached to the arm of the biomicroscope.

Dr. Busacca's book is primarily the work of a histologist who applies his microscopic knowledge to the study of gonioscopy. He used especially the narrow slit of the biomicroscope in optical section to determine the arrangement of the structures in depth, and he thinks that a strong light is confusing and dazzling to the observer. He asserts that, although using better instruments, modern authors "have not added anything to the observations of Salzmann with the ophthalmoscope" and "entirely ignore structural details which this author pointed out."

This assertion is not accurate. Probably Dr. Busacca has studied the angle particularly in the articles of former observers, such as Virchow, for the histologic characteristics, and Salzmann, for clinical details. He entirely ignores the work of modern authors; for instance, he dismisses with two or three words all the research on comparative anatomy of the angle which I have published and which throws great light on the architectural building of the structures of the chamber recess.

The first part of his book is devoted to the histology of the angle. Although he criticizes the authors who have "a mania for creating new names," he immediately proceeds to give new ones to almost all the organs and tissues seen in the gonioscopic field. Starting with the Schwalbe line, which he calls the "anterior limiting white line," and the scleral spur, which he calls the "white line of the spur," he describes the band of the ciliary body as "the shadow of the muscle." This entirely inappropriate name is meant to convey to the observer the fact that the fibers of the ciliary muscle are present behind the surface of the ciliary body. He asserts that the "shadow" of the circular bundles of the muscle can be seen with a narrow slit. To increase the confusion, he divides the "muscular shadow" in two bands, which are of different color; the anterior band, near the spur, is of a darker gray

hue, and he calls it "the suprachoroidal shadow." This color is determined, in his opinion, by the pigment present at this place on the inner surface of the sclera and between the longitudinal fibers of the muscle. The posterior band is a lighter brown. He believes that the muscle fibers are "probably" red, although it is known that smooth muscles are usually gray white.

Studying the iris, he describes cases in which the most prominent ridge in the membrane, instead of being near and over the sinus of the chamber, is placed a little forward on the surface of the iris. He gives the name "iris groove" to the part of the surface of the iris behind the most prominent ridge. The designation is inaccurate, as there is no groove at this place—only the flat surface of the iris, which runs directly outward, to attach itself to the anterior surface of the ciliary body.

He calls the "basal band of the iris processes" the part of the root of the iris to which are inserted the so-called iris processes. I have shown conclusively by studies in comparative anatomy that these "iris processes" are the rudimentary fibers of the pectinate ligament, so strongly developed in lower mammals. These fibers have also been described as the "uveal trabecula." Busacca gives them three different names. Sometimes he calls them "iris processes"; at other times the "uveal trabecula" and at still others the "iris trabecula." In his color scheme of the structures of the angle, he shows the pectinate fibers as arising from almost one third of the peripheral surface of the iris. This is contrary to all my microanatomic observations. It appears that he made this drawing partly from anatomic specimens. He probably stretched the iris so much that the root became longer and the pectinate fibers advanced forward.

An anatomic characteristic described by Busacca is worth while retaining. The band of the ciliary body is covered with a semitranslucent membrane, which he calls the "trabeculoconnective lamella." This is made by the lower portion of the corneal trabecula, which extends beyond and back from the scleral spur. The fibers of the meshwork are inserted into a connective tissue layer, which covers the ciliary muscle and is continuous in depth with the septums that divide the muscular bundles. This lamella follows the concave surface of the sinus, from the spur to the root of the iris. It has already been described by other authors (Virchow, Fuchs and Lauber), but the name "trabeculoconnective lamella" given by Busacca is worth keeping, as it calls attention to the relationship of the fibers of the trabecula to the surface of the ciliary body. This is important in pathologic conditions, especially in cases of simple glaucoma.

The clinical chapters of Busacca's book are more important. His description of the various kinds of peripheral synechias (he calls them "goniosynechias") is interesting and throws light on the fission of the anterior mesodermal layer of the iris in cases of certain adhesions. He considers the synechia the result "of an inflammatory process localized on the sinus wall."

In the chapter on glaucoma, he states that gonioscopy has not been able to produce a "pathognomonic sign characteristic of this disease" and expresses the belief that the problem cannot be solved by this method. No one has claimed that the pathogenesis of glaucoma can be elucidated by gonioscopy alone. However, Busacca ignores the results of

hundreds of observations made, in order of sequence, by myself, Werner, Sugar and Gradle, Barkan, McLean, Kronfeld and others, which have conclusively shown that there is a great difference in the appearance of the angle in cases of simple glaucoma and in those of the congestive type. We have shown that in cases of the former the angle is almost always open, while in cases of the latter the angle is narrow and usually peripheral synechias are present. Busacca, on the contrary, says that in the great number of cases of simple glaucoma he has studied the iridocorneal sinus could be classified as a "narrow sinus" because the "line of the crests" (the most prominent ridge of the iris) was facing the anterior part of the trabecula or the cornea, "giving to the broad slit the impression of a wide sinus on account of the transparency of the corneal tissue." However, he has also observed cases of wide sinuses. In cases of acute congestive glaucoma, he states that the narrow entrance to the sinus has no direct bearing on the glaucomatous attack and that it is not a predisposing cause. This is contrary to the opinion expressed by many authors, from Fuchs to Gradle and Sugar. Busacca still clings to the opinion of Salzmann, who stated the belief that atropine enlarges the chamber sinus and physostigmine narrows it. Sugar has shown that the converse is true.

The chapters on gonioscopy in cases of inflammatory processes of the uvea and of disease of the cornea are well written and informative. I shall mention especially those relating to the gonioscopic aspects after operations on the eyeball and the chapter on congenital malformations of the iris.

The book ends with a chapter on "Experimental Gonioscopy," in which the author studies eyes which have undergone iridectomy for cataract, glaucoma or other diseases. He uses an ocular micrometer attached to the eyepiece of the slit lamp, and with the narrow slit he examines the anterior part of the ciliary body and the ciliary processes visible in the area of the coloboma, noting the number of the processes and their shape, position and dimensions. He then instills atropine or physostigmine into the eye and observes the movements of the heads of the processes. He has seen with use of atropine that the heads retract and the edge of the lens (if it was previously covered) becomes exposed and the sinus shallower. Physostigmine, on the contrary, produces an enlargement of the processes. These cover the margin of the lens, and the entrance to the sinus narrows and becomes deeper.

It is unfortunate that the constant use of new names makes Busacca's descriptions confused and complicated. However, he deserves great credit for his painstaking work and for enlarging and completing the chapter on biomicroscopy of the structures of the angle first undertaken by Bangerter and Goldman. MANUEL URIBE TRONCOSO.

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President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

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Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

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Executive Secretaries: Dr. Conrad Berens, 35 E. 70th St., New York. Dr. M. E. Alvaro, 1511 Rua Consolacão, São Paulo, Brazil.
Place: Habana, Cuba. Time: February 1948.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

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Ophthalmological Society of Australia

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Secretary: Dr. D. A. Williams, 27 Commonwealth St., Sydney.
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Place: London. Time: May 30-June 1-2, 1946.

Ophthalmology Society of Bombay

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Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Oxford Ophthalmological Congress

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Place: Rosario. Time: Last Saturday of every month, April to November. All correspondence should be addressed to the President.

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SECTIONAL

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Secretary-Treasurer: Dr. W. H. Turnley, 1 Atlantic St., Stamford, Conn.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. William O. Martin Jr., Doctors Bldg., Atlanta.
Secretary-Treasurer: Dr. C. K. McLaughlin, 666 Cherry St., Macon.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. W. E. Stewart, 721 Wabash Ave., Terre Haute.
Secretary: Dr. Russell A. Sage, 23 E. Ohio St., Indianapolis.
Place: French Lick. Time: First Wednesday in April.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. S. A. O'Brien, 1 N. Federal Ave., Mason City.

KANSAS STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. B. Granger, Emporia.
Secretary: Dr. George F. Gsell, 911 Beacon Bldg., Wichita 2.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. George S. Adkins, 121 N. President St., Jackson, Miss.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

Medical Society of the State of Pennsylvania, Section on Eye, Ear, Nose and Throat Diseases

Chairman: Dr. William T. Hunt Jr., 1205 Spruce St., Philadelphia 7.
Secretary: Dr. Gabriel Tucker, 250 S. 18th St., Philadelphia 3.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Edmond L. Cooper, 1553 Woodward Ave., Detroit 26.
Secretary: Dr. Ralph H. Gilbert, 110 Fulton St. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Otolaryngology

President: Dr. Karl C. Wold, 1051 Lowry Bldg., St. Paul 2.
Secretary: Dr. William A. Kennedy, 372 St. Peter St., St. Paul 2.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. H. Casebeer, 44 W. Park Ave., Butte.
Secretary: Dr. Fritz D. Hurd, 309 Medical Arts Bldg., Great Falls.

Nebraska Academy of Ophthalmology and Otolaryngology

President: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. John Peterson, 1307 N St., Lincoln.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. C. W. Buvinger, 50 Washington St., East Orange.
Secretary: Dr. Z. Laurence Griesemer, 1145 E. Jersey St., Elizabeth.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Maxwell D. Ryan, 660 Madison Ave., New York 21.
Secretary: Dr. Thomas H. Johnson, 30 W. 59th St., New York.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. A. J. Ellington, 412 S. Spring St., Burlington.
Secretary: Dr. J. A. Harrill, Bowman Gray School of Medicine, Winston-Salem.
Place: Hendersonville. Time: Sept. 16-19, 1946.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. E. D. Perrin, 221–5th St., Bismarck.
Secretary-Treasurer: Dr. M. T. Lampert, Minot.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. Wilfred Belnap, 833 S. W. 11th Ave., Portland.
Secretary-Treasurer: Dr. C. W. Kuhn, 1020 S. W. Taylor St., Portland 5.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

Pennsylvania Academy of Ophthalmology and Otolaryngology

President: Dr. Thomas F. Furlong Jr., 36 Parking Plaza, Ardmore.
Secretary: Dr. Benjamin F. Souders, 143 N. 6th St., Reading.
Time: Last week in April.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society, Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. J. H. Stokes, 125 W. Cheves St., Florence.
Secretary-Treasurer: Dr. Roderick Macdonald, 330 E. Main St., Rock Hill.
Place: Hendersonville, N. C. Time: Sept. 16-19, 1946.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. George Burchfield, Maryville.
Secretary-Treasurer: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. F. H. Rosebrough, 603 Navarro St., San Antonio.
Secretary: Dr. M. K. McCullough, 1717 Pacific Ave., Dallas.

Utah Ophthalmological Society

President: Dr. E. B. Fairbanks, 315 Medical Arts Bldg., Salt Lake City.
Secretary-Treasurer: Dr. Dean Spear, 516 Boston Bldg., Salt Lake City.
Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Thomas E. Hughes, 1000 W. Grace St., Richmond.
Secretary-Treasurer: Dr. Francis H. McGovern, 105 S. Union St., Danville.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. E. L. Mather, 39 S. Main St., Akron, Ohio.
Secretary-Treasurer: Dr. V. C. Malloy, 2d National Bank Bldg., Akron, Ohio.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. B. M. Cline, 153 Peachtree St. N. E., Atlanta, Ga.
Secretary: Dr. Lester A. Brown, 815 Doctors Bldg., Atlanta, Ga.
Place: Academy of Medicine. Time: 7:30 p. m., fourth Monday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Jonas Friedenwald, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Fred Reese, 330 N. Charles St., Baltimore 1.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to March.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. W. Chunn Parsons, 425 Woodward Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. Benjamin C. Rosenthal, 140 New York Ave., Brooklyn.
Secretary-Treasurer: Dr. Louis Freimark, 256 Rochester Ave., Brooklyn 13.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. William H. Howard, 389 Linwood Ave., Buffalo 9.
Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo 9.
Time: Second Thursday of each month from October to May.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. Douglas Chamberlain, Chattanooga Bank Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. W. A. Mann, 30 N. Michigan Ave., Chicago 2.
Secretary: Dr. J. R. Fitzgerald, 3215 W. North Ave., Chicago.
Place: Continental Hotel, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati General Hospital Ophthalmology Staff

Chairman: Dr. D. T. Vail, 441 Vine St., Cincinnati.
Secretary: Dr. A. A. Levin, 441 Vine St., Cincinnati.
Place: Cincinnati General Hospital. Time: 7:45 p. m., third Friday of each month except June, July and August.

Cleveland Ophthalmological Club

Chairman: Dr. M. Paul Motto, Rose Bldg., Cleveland.
Secretary: Dr. H. H. Wygand, 624 Guardian Bldg., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Burton Chance, 317 S. 15th St., Philadelphia.
Clerk: Dr. George F. J. Kelly, 37 S. 20th St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. M. Goldberg, 328 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. W. J. Miller, 21 E. State St., Columbus, Ohio.
Place: University Club. Time: 6:15 p. m., first Monday of each month, from October to May, inclusive.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. L. W. O. Janssen, 710 Medical Professional Bldg., Corpus Christi, Texas.
Secretary: Dr. F. B. Kelly, 519 Medical Professional Bldg., Corpus Christi, Texas.
Time: 6:30 p. m., third Tuesday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Speight Jenkins, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. L. Darrough, Dallas Medical and Surgical Clinics, Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. H. I. McPherrin, 406–6th Ave., Des Moines, Iowa.
Secretary-Treasurer: Dr. C. C. Jones, Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., fourth Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Wesley G. Reid, 974 Fisher Bldg., Detroit 2.
Place: Club rooms of Wayne County Medical Society. Time: First Monday of each month, November to April, inclusive.

Detroit Ophthalmological Society

President: Dr. Bruce Fralick, 201 S. Main St., Ann Arbor, Mich.
Secretary: Dr. William S. Gonne, 619 David Whitney Bldg., Detroit 26.
Place: Club rooms of Wayne County Medical Society. Time: 6:30 p. m., third Thursday of each month from November to April, inclusive.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. Frank C. Furlong, 713 Union St., Schenectady.
Secretary-Treasurer: Dr. E. Martin Freund, 762 Madison Ave., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. C. R. Lees, 602 W. 10th St., Fort Worth 2, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1305 Medical Arts Bldg., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Ophthalmological and Oto-Laryngological Section

President: Dr. J. Matt Robison, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. John H. Barrett, 1304 Walker Ave., Houston, Texas.
Place: River Oaks Country Club. Time: 6:30 p. m., second Thursday of each month from October to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. J. Jerome Littell, 603 Hume Mansur Bldg., Indianapolis.
Secretary: Dr. J. Lawrence Sims, 303 Hume Mansur Bldg., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Edgar Johnson, 906 Grand Ave., Kansas City, Mo.
Secretary: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Francis Carl Hertzog, 117 E. 8th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Robert G. Thornburgh, 117 E. 8th St., Long Beach, Calif.
Place: Seaside Hospital. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. A. R. Robbins, 930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. K. C. Brandenburg, 110 Pine Ave., Long Beach 2, Calif.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye and Ear Society

President: Dr. Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. W. Fish, 321 W. Broadway, Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Lower Anthracite Eye, Ear, Nose and Throat Society

Chairman: Each member in alphabetical order.
Secretary: Dr. James J. Monohan, 31 S. Jardin St., Shenandoah, Pa.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. P. S. Constantinople, 1835 I St. N. W., Washington.
Secretary: Dr. Frazier Williams, 1801 I St. N. W., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month from September to May.

Milwaukee Oto-Ophthalmic Society

President: Dr. Ralph T. Rank, 238 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. Frank G. Treskow, 411 E. Mason St., Milwaukee 2.
Place: University Club. Time: 6:30 p. m., fourth Tuesday of each month from October to May.

Montgomery County Medical Society

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. J. Rosenbaum, 1396 Ste. Catherine St. W., Montreal, Canada.
Secretary: Dr. L. Tessier, 1230 St. Joseph Blvd. E., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. M. M. Cullom, 700 Church St., Nashville, Tenn.
Secretary: Dr. R. E. Sullivan, 432 Doctors Bldg., Nashville 3, Tenn.
Place: James Robertson Hotel. Time: 6:30 p. m., third Monday of each month from October to May.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Secretary: Dr. Mercer G. Lynch, 1018 Maison Blanche Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to May.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Brittain F. Payne, 17 E. 72d St., New York 21.
Secretary: Dr. Milton Berliner, 57 W. 57th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

President: Dr. Benjamin Friedman, 6 W. 77th St., New York.
Secretary: Dr. Benjamin Esterman, 983 Park Ave., New York 28.
Place: New York Academy of Medicine, 2 E. 103d St. Time: 8 p. m., first Monday of each month from October to May, inclusive.

OKLAHOMA CITY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. S. R. Shaver, 117 N. Broadway, Oklahoma City.
Secretary: Dr. William Mussil, Medical Arts Bldg., Oklohoma City.
Place: University Hospital. Time: Second Tuesday of each month from September to May.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. A. A. Steinberg, 1502 Farnam St., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha 2.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m. dinner; 7 p. m. program; third Wednesday of each month from October to May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. Thomas Sanfacon, 340 Park Ave., Paterson, N. J.
Secretary-Treasurer: Dr. J. Averbach, 435 Clifton Ave., Clifton, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

President: Dr. Isaac Tassman, 136 S. 16th St., Philadelphia.
Secretary: Dr. Glen Gregory Gibson, 255 S. 17th St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Jay G. Linn, Jenkins Arcade, Pittsburgh.
Secretary: Dr. Robert J. Billings, Jenkins Arcade, Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

READING EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Claude W. Bankes, 212 N. 6th St., Reading, Pa.
Secretary: Dr. Paul C. Craig, 232 N. 5th St., Reading, Pa.
Place: Wyomissing Club. Time: 6:30 p. m., third Wednesday of each month from September to July.

Richmond Eye, Ear, Nose and Throat Society

President: Dr. Luther C. Brawner, Professional Bldg., Richmond, Va.
Secretary: Dr. Clifford A. Folkes, Professional Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. Frank Barber, 75 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.

St. Louis Ophthalmic Society

President: Dr. A. Lange, 3903a Olive St., St. Louis.
Secretary: Dr. William Kleinberg, Frisco Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Fourth Friday of each month from October to April, inclusive, except December, at 8:00 p. m.

San Antonio Ophthalmo-Oto-Laryngological Society

President: Dr. Belvin Pritchett, 705 E. Houston St., San Antonio 5, Texas.
Secretary-Treasurer: Lt. Col. John L. Matthews, AAF School of Aviation Medicine, Randolph Field, Texas.
Place: San Antonio, Brooke General Hospital, Randolph Field or San Antonio Aviation Cadet Center. Time: 7 p. m., second Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. C. B. Cowan, 490 Post St., San Francisco.
Secretary: Dr. D. Harrington, 384 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.
Secretary-Treasurer: Dr. Kenneth Jones, Medical Arts Bldg., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Clarence A. Veasey Sr., 421 W. Riverside Ave., Spokane, Wash.
Secretary: Dr. Clarence A. Veasey, 421 W. Riverside Ave., Spokane, Wash.
Place: Spokane Medical Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. A. H. Rubenstein, 713 E. Genesee St., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. H. Blaisdell, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toledo Eye, Ear, Nose and Throat Society

Chairman: Dr. W. W. Randolph, 1838 Parkwood Ave., Toledo 2, Ohio.
Secretary: Dr. John L. Roberts, 316 Michigan St., Toledo, Ohio.
Place: Toledo Club. Time: Each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. R. G. C. Kelly, 14 Lynwood Ave., Toronto 5, Canada.
Secretary: Dr. Alfred Elliott, 802 Medical Arts Bldg., Toronto 5, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. Harold M. Downey, 1740 M St. N. W., Washington, D. C.
Secretary-Treasurer: Dr. Richard W. Wilkinson, 1408 L St. N. W., Washington, D. C.
Place: Medical Society of District of Columbia Bldg., 1718 M St. N. W., Washington, D. C. Time: 7:30 p. m., first Monday in November, January, March and May.

Wilkes-Barre Ophthalmological Society

Chairman: Each member in turn.
Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.
Place: Office of chairman. Time: Last Tuesday of each month from October to May.

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